MISSOURI DEPARTMENT OF
HEALTH

John Ashcroft
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North End Site
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July 7, 1992

Mr. Robert L. Morby
Chief, Superfund Branch
Waste Management Division
U. S. Environmental Protection Agency
726 Minnesota Avenue
Kansas City, Kansas 66101

Dear ~~Mr. Morby~~ Bob:

Enclosed please find a revised copy of the draft Final Residual Risk Assessment for Groundwater Contamination, North End Site, Kansas City, Missouri. This document will replace one which was sent to you on July 6, 1992. Some typographical and formatting errors were discovered and these have been corrected.

The Missouri Department of Health apologizes for any inconvenience this may have caused. If you have any questions or additional comments, please feel free to call Ms. Cherri Baysinger-Daniel or Mr. Chuck Arnold at (314) 751-6102.

Sincerely,

Daryl W. Roberts
Chief
Bureau of Environmental Epidemiology

DWR:CCA

DRAFT FINAL RESIDUAL RISK ASSESSMENT FOR GROUNDWATER CONTAMINATION NORTH END SITE

1.0 INTRODUCTION

1.1 Objective

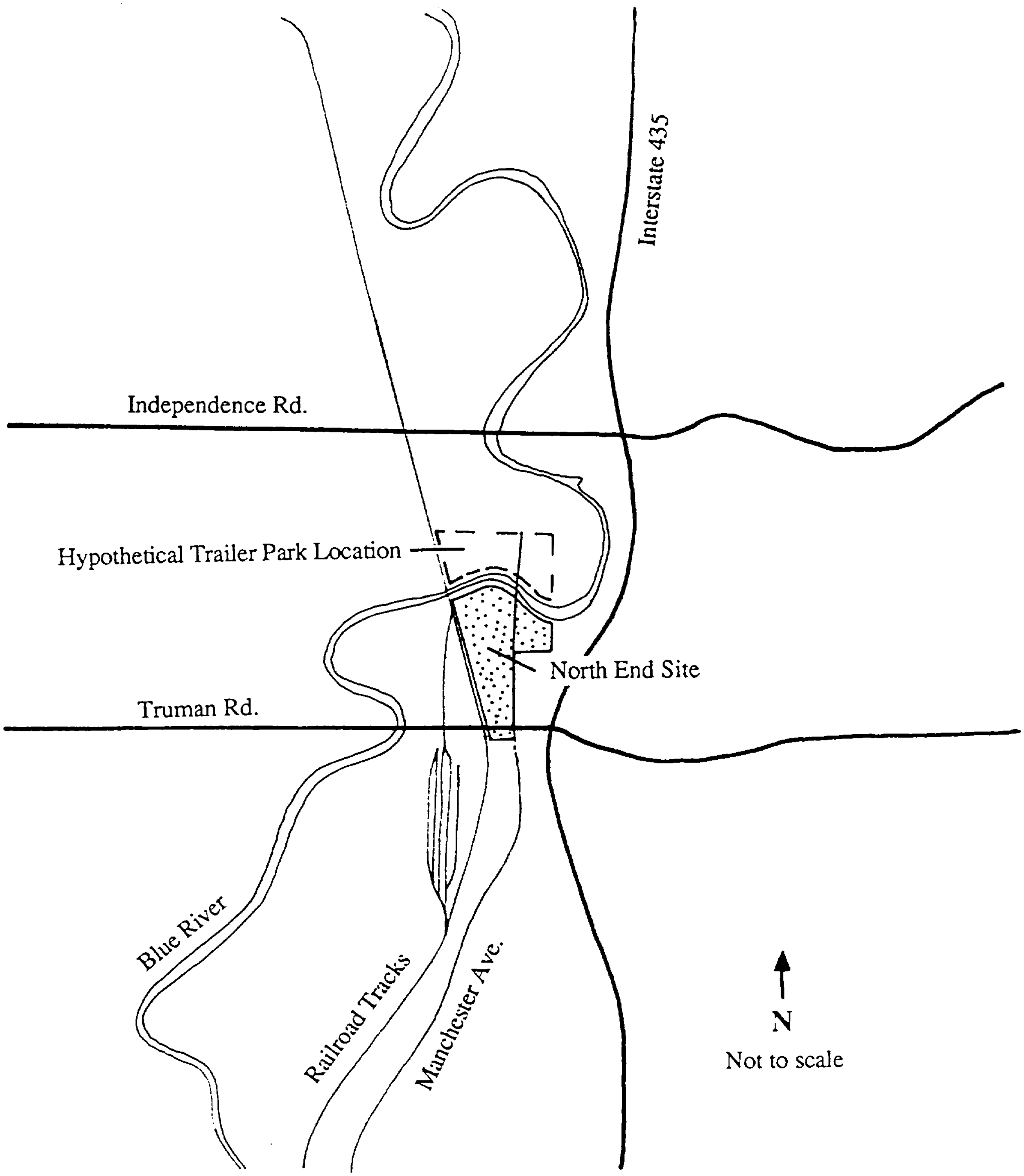
The North End Site was used for the disposal for lead-contaminated wastes resulting from the production of steel. Site cleanup included soil removal and the installation and sampling of groundwater monitoring wells. Residual groundwater contamination was detected after the primary contaminant source had been removed. The Missouri Department of Health (DOH) was tasked by the U. S. Environmental Protection Agency (EPA) to conduct a risk assessment to determine if residual groundwater contamination warrants a cleanup of groundwater at the North End Site.

1.2 Site Background

The North End Site is located in an industrialized area of Kansas City, Missouri, within the Armco complex, an active steel manufacturing facility (Figure 1). From 1962 to 1980, the North End Site was used as a landfill for the disposal of lead-contaminated electric furnace baghouse dusts generated during steel production.

In complying with an Administrative Order of Consent (AOC), Armco Inc. conducted a removal of lead-contaminated soil at the North End Site. This included excavation, sorting and staging, testing, off-site disposal and verification of the effectiveness of the removal action. Over 26,000 cubic yards of material were excavated and disposed of in permitted landfills. Lead concentrations in soil samples taken following completion of soil removal were less than the clean-up criterion of 238 mg/kg total lead. After confirmatory sampling and analysis was completed, the site was backfilled, graded, and seeded.

Pursuant to the AOC, Armco installed two deep and three shallow monitoring wells (in addition to ten existing wells) for further collection and analysis of groundwater. Sampling of all site wells was conducted in March and again in May, 1991. Lead and several volatile organic compounds (VOCs) were detected.



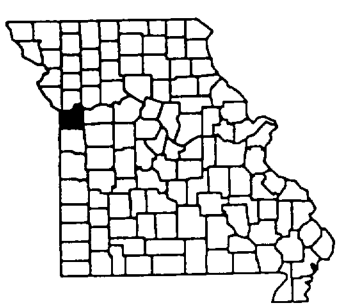


Figure 1.
General Site Diagram and Location of Hypothetical Trailer Park
North End Superfund Site
Kansas City, MO.

1.3 Scope of Risk Assessment

This risk assessment will evaluate the human health risks posed to a hypothetical future off-site resident drinking and showering with groundwater contaminated by lead and the volatile organic compounds detected at the North End Site.

2.0 IDENTIFICATION OF CHEMICALS OF POTENTIAL CONCERN

2.1 Site Geology

The North End Site is underlain by Quaternary Alluvium of the Blue River. Soil consistency ranges from moist to saturated interbedded clayey silt and silty clay with small amounts of fine sand in the upper soils to poorly sorted silty gravels and sand in the lower soils sitting above bedrock. A zone of reduced permeability consisting of stiff, silty clay lies between the two soil types. Groundwater investigation results indicate the unconsolidated aquifer is comprised of an upper and lower zone each having slightly different hydraulic conductivities. Sampling results (absence of VOCs in deep wells) suggest the alluvium acts as two separate aquifers, both discharging to the Blue River.

2.2 Data Collection

Prior to the removal action, ten shallow monitoring wells (screened between 16 to 25 feet below ground surface (bgs)) were located on the North End Site. Under the AOC, Armco installed five additional wells; two deep wells (screened between 58 to 64 feet bgs) and three shallow wells (screened between 8 to 13 feet bgs). Because of deep excavation for waste removal prior to the May sampling, one well was abandoned and sealed. A replacement well was installed in close proximity. Fifteen wells were sampled during March and May of 1991, and the results used in this risk assessment.

2.3 Data Evaluation

All groundwater samples were analyzed for total phenolics, nitrates, total and dissolved lead, priority pollutant VOCs, polychlorinated biphenyls, pH, specific conductance and alkalinity. Total lead was present in detectable levels in all groundwater samples in concentrations up to 400 ug/L (Appendix I). Dissolved lead was not detected in any groundwater samples. The following VOCs were detected at least once in at least one well during groundwater sampling: chloroethane, chloromethane, 1,2 dichlorobenzene,

1,1 dichloroethane, 1,2 dichloroethane, 1,1 dichloroethene, 1,2 dichloroethene, tetrachloroethene, 1,1,1 trichloroethane, trichloroethene, and vinyl chloride (Appendix I).

Contaminants of concern were limited to total lead and the volatile organic compounds detected at least once during groundwater sampling (Table 1). Average contaminant concentrations for each well were calculated from the March and May sampling data. For contaminants of concern undetected during one sampling round, one-half the detection limit was used to calculate average contaminant concentrations (standard practice in Superfund risk assessment). Contaminant concentrations for VOCs in wells from which a replicate sample had been taken are the arithmetic average of the initial and replicate sample results. Contaminant concentrations from all wells were combined to calculate the site mean, maximum, minimum, standard deviation and a 95% Upper Confidence Limit of the mean value (UCL) values (Table 1).

2.4 Uncertainties

During the second round of sampling, vinyl chloride was detected in only one sample; the compound identification was certain but the concentration was an estimated value (J-qualified, Appendix I). Chloromethane, 1,2 dichlorobenzene, and 1,2 dichloroethene were detected during the first round of sampling but not detected during the second round of sampling (Appendix 1). Because these compounds were included in the risk assessment, the true risk posed by the site may be overestimated.

3.0 EXPOSURE ASSESSMENT

3.1 Current Exposure Pathways

Land use in the vicinity of the North End Site is currently industrial. Contaminated soil was removed, precluding current or future exposure to contaminated soil. No drinking water wells are currently located on the North End property, thus on-site exposure through ingestion of contaminated groundwater is not expected. Because the risk to human health from current exposures via groundwater is essentially zero,.no current exposure pathways were evaluated.

3.2 Future Exposure Pathways

The industrialized nature of the area is not expected to change, but the possibility exists for a residential community to be established across the Blue River north of the site (see Figure 1). For the purposes of this risk assessment, EPA has directed DOH to assume

Table 1.

Summary Statistics for Contaminants of Concern in Groundwater at the North End Site, Kansas City, MO.

Contaminant	Mean Concentration (mg/L)	Maximum Value (mg/L)	Minimum Value (mg/L)	Standard Deviation	95% Upper Confidence Limit
Total Lead	0.063	0.229	0.0095	0.055	0.0918
Chloroethane	0.017	0.170	0.005	0.041	0.0390
Chloromethane	0.007	0.025	0.005	0.005	0.00972
1,2 Dichlorobenzene	0.004	0.013	0.0025	0.003	0.00521
1,1 Dichloroethane	0.065	0.610	0.0025	0.159	0.149
1,2 Dichloroethane	0.003	0.013	0.0025	0.003	0.00481
1,1 Dichloroethene	0.039	0.340	0.0025	0.086	0.0840
1,2 Dichloroethene	0.004	0.018	0.00225	0.004	0.00580
Tetrachloroethene	0.005	0.029	0.0025	0.007	0.00855
1,1,1 Trichloroethane	0.028	0.220	0.0025	0.057	0.0578
Trichloroethene	0.004	0.013	0.0025	0.003	0.00569
Vinyl Chloride	0.008	0.035	0.005	0.008	0.0119

future land use on adjacent property would be residential; i.e., land across the Blue River from the North End Site could be used as a trailer park and drinking water would be supplied by a community well. Exposure pathways for future residential land use were ingestion of contaminated drinking water and inhalation of volatilized VOCs during showering.

The Blue River is generally considered to be a hydrologic barrier to groundwater movement. For the purposes of this risk assessment and to ensure protectiveness of human health, it is assumed that the community well would be large enough to pull water across the hydrologic divide formed by the Blue River. EPA estimates that the well would obtain at most 1% of its water supply from the North End Site, thus contaminant concentrations in the community well would be 1/100th of the average concentrations beneath the North End site (Table 2). EPA believes that this is conservative and tends to overestimate risk.

3.3 Reasonable Maximum Exposures (RMEs)

Pursuant to the National Contingency Plan (40 CFR, Part 300), EPA estimates human health risk for a reasonable maximum exposure (RME). For the future land use scenario, two RMEs were developed by EPA using site specific assumptions. RME 1 was a 15-kilogram child, 0-6 years of age, living in the trailer park for 6 years, ingesting and showering with contaminated groundwater. RME 2 was a 70-kilogram adult living in the trailer park for thirty years ingesting contaminated groundwater and inhaling volatilized VOCs while showering.

3.4 Calculation of Air Concentrations

Contaminant concentrations in air (mg/m^3) while showering (Table 3) were calculated from groundwater concentrations (mg/L) using the following formula:

$$\text{Concentration in Air} = \frac{(\text{Concentration in Water})(\text{Liters/shower})(\text{Volatilization Factor})}{(\text{Room Volume})}$$

This formula was modified from a formula provided by EPA's Environmental Criteria and Assessment Office (ECAO 1991). In these calculations, the values 180 liters and 10 m^3, respectively, were used as site-specific estimates of the number of liters per shower and the room volume. A value of 0.5 (0.0005 x 1000 L/m^3) was used as the volatilization factor (EPA 1991c).

Table 2.

Calculated Concentrations of Contaminants of Concern in a Hypothetical Future Drinking Water Well, North End Site, Kansas City, MO.

Contaminant	95% Upper Confidence Limit (mg/L)	Assumed Percentage of Contaminated Water Entering the Community Well	Calculated Concentration in Community Well Water (mg/L)
Total Lead	9.18 x 10-2	1 %	9.18×10^{-4}
Chloroethane	3.90 x 10-2	1 %	3.90×10^{-4}
Chloromethane	9.72 x 10-3	1 %	9.72×10^{-5}
1,2 Dichlorobenzene	5.21×10^{-3}	1 %	5.21×10^{-5}
1,1 Dichloroethane	1.49×10^{-1}	1 %	1.49×10^{-3}
1,2 Dichloroethane	4.81×10^{-3}	1 %	4.81×10^{-5}
1,1 Dichloroethene	8.40×10^{-2}	1 %	8.40×10^{-4}
1,2 Dichloroethene	5.80×10^{-3}	1 %	5.80×10^{-5}
Tetrachloroethene	8.55×10^{-3}	1 %	8.55×10^{-5}
1,1,1 Trichloroethane	5.78×10^{-2}	1 %	5.78×10^{-4}
Trichloroethene	5.69×10^{-3}	1 %	5.69×10^{-5}
Vinyl Chloride	1.19×10^{-2}	1 %	1.19×10^{-4}

Table 3.

Calculated Concentrations of Volatile Compounds in Air While Showering North End Site, Kansas City, MO.

Contaminant	Size of Bathroom (m3)	Liters of Water Used Per Shower (L)	Contaminant Concentration in Groundwater (mg/L)	Volatilization Factor	Contaminant Concentration in Air (mg/m^3)
Chloroethane	10	180	0.00039	0.5	0.00351
Chloromethane	10	180	0.0000972	0.5	0.0008748
1,2 Dichlorobenzene	10	180	0.0000521	0.5	0.0004689
1,1 Dichloroethane	10	180	0.00149	0.5	0.01341
1,2 Dichloroethane	10	180	0.0000481	0.5	0.0004329
1,1 Dichloroethene	10	180	0.00084	0.5	0.00756
1,2 Dichloroethene	10	180	0.000058	0.5	0.000522
Tetrachloroethene	10	180	0.0000855	0.5	0.0007695
1,1,1 Trichloroethane	10	180	0.000578	0.5	0.005202
Trichloroethene	10	180	0.0000569	0.5	0.0005121
Vinyl Chloride	10	180	0.000116	0.5	0.001044

3.5 Estimation of Chemical Intakes

Intake rates for all contaminants except lead were quantified using the pathway-specific equations (Tables 4 and 5) taken from EPA (1989) Risk Assessment Guidance for Superfund: Volume I (RAGS). Exposure variables used in the equations were chosen by EPA so that the combination of all intake variables resulted in a RME for each contaminant within a pathway (Appendix II).

The Lead Biokinetic Uptake Model was used to estimate intake of lead from groundwater at this site. The model was run using a groundwater concentration of 0.000918 mg/L with default values for soil, air, food and paint.

4.0 TOXICITY ASSESSMENT

4.1 Noncarcinogenic Effects

Reference Doses (RfDs) and Reference Concentrations (RfCs) are the toxicity values used in assessing noncarcinogenic effects from oral and inhalation exposure, respectively. EPA's Integrated Risk Information System (IRIS) contains contaminant specific RfD and RfC values which have been verified by an intra-Agency work group. RfD and RfC values which have not been verified may be found in EPA Health Effects Assessment Summary Tables (HEAST, EPA 1991b). Available toxicity values and effects of concern associated with exposure to specific contaminants are summarized in Table 6.

Unit Risks were converted to RfCs using the formula taken from the preface to HEAST, 1991 Annual Volume (EPA 1991). The formula is as follows:

$$RfC = (\text{Unit Risk})\ (\text{Inhalation Rate})\ (\text{Body Weight})$$

where Inhalation Rate = 0.83 m^3/hour and Body Weight = 70 kg.

Currently, there are no toxicity values for lead in IRIS or HEAST (EPA 1991b). Lead intake affects virtually every system in the body. Among the most serious effects of lead exposure are the central nervous system effects seen in young children. These effects range from impaired learning ability and a decrease in IQ scores to brain damage. Other effects are a decrease in growth of children, a decrease in hearing acuity and adverse effects on the kidneys and hematopoietic systems (CDC 1991). To assess the adverse health effects of lead exposure, EPA currently advises use of the Lead Biokinetic Uptake Model. This model combines intake variables from several potential lead exposure pathways and

Table 4.

Intake Equations for Ingestion of Contaminated Groundwater North End Site, Kansas City, MO.*

Equation:

Chronic Daily Intake (mg/kg/day)=CW x IR x EF x ED / (BW x AT)

Where:

CW=Chemical Concentration in Groundwater (mg/L)
IR=Ingestion Rate (L water/day)
EF=Exposure Frequency (days/year)
ED=Exposure Duration (years)
BW=Body Weight (kg)
AT=Averaging Time (days)

Variable values:

CS=site specific calculated value (Table 2)
IR=1 L/day - child
2 L/day - adult (EPA 1990)
EF=365 days/year (number of days in a year)
ED=6 years - child
30 years - adult
BW=15 kg (arithmetic mean of 50^{th} percentile body weights of children aged 0-6 years)
70 kg - adult (EPA 1990)
AT=2190 days for child - noncarcinogenic effects (ED x 365 days/year)
10950 days for adult - noncarcinogenic effects (ED x 365 days/year)
25550 days for child and adult carcinogenic effects (70 years x 365 days/year)

*Formula was obtained from EPA 1989

Table 5.

Inhalation of Volatilized Compounds While Showering
North End Site, Kansas City, MO.*

Equation:

Chronic Daily Intake (mg/kg/day)=CA x IR x ET x EF x ED / (BW x AT)

Where:

CA=Chemical Concentration in air (mg/m^3)
IR=Inhalation Rate (m^3/hr)
ET=Exposure Time (hours/day)
EF=Exposure Frequency (days/year)
ED=Exposure Duration (years)
BW=Body Weight (kg)
AT=Averaging Time (days)

Variable values:

CA=calculated chemical concentration (Table 3)
IR=0.83 m^3/hour (EPA 1990)
ET=0.25 hour/day (site specific estimate)
EF=365 days/year (assumes one shower per day)
ED=6 years - child
30 years - adult
BW=15 kg (arithmetic mean of 50th percentile body weights of children aged 0-6 years)
70 kg - adult (EPA 1990)
AT=2190 days for child - noncarcinogenic effects (ED x 365 days/year)
10950 days for adult - noncarcinogenic effects (ED x 365 days/year)
25550 days for child and adult carcinogenic effects (70 years x 365 days/year)

*Formula was obtained from EPA 1989

Table 6.

Noncarcinogenic Toxicity Information for Chemicals of Concern at the North End Site, Kansas City, MO.*

Compound	Oral Reference Dose (mg/kg/day)	Inhalation Reference Dose (mg/kg/day)	Effects of Concern (oral; inhalation)
Chronic Exposures			
Chloroethane	ND	1×10^{-1}	NA; developmental toxicity
1,2 Dichlorobenzene	9×10^{-2}	4×10^{-2}	Liver effects; decreased body weight gain
1,1 Dichloroethane	1×10^{-1}	1×10^{-1}	NA; kidney damage
1,1 Dichloroethene	9×10^{-3}	ND	Liver lesions; NA
1,2 Dichloroethene	1×10^{-2}	ND	Decreased hematocrit and hemoglobin; NA
Tetrachloroethene	1×10^{-2}	ND	Hepatotoxicity; NA
1,1,1 Trichloroethane	9×10^{-2}	3×10^{-1}	Hepatotoxicity, hepatotoxicity
Trichloroethene**	6 x 10-3	ND	Kidney and liver effects; NA
Subchronic Exposures			
Chloroethane	ND	1×10^{1}	NA; developmental toxicity
1,2 Dichlorobenzene	9×10^{-1}	4×10^{-1}	Liver effects; decreased body weight gain
1,1 Dichloroethane	1×10^{0}	1×10^{0}	NA; kidney damage
1,1 Dichloroethene	9×10^{-3}	ND	Liver lesions; NA
1,2 Dichloroethene	1×10^{-1}	ND	Decreased hematocrit and hemoglobin; NA
Tetrachloroethene	1×10^{-1}	ND	Hepatotoxicity; NA
1,1,1 Trichloroethane	9×10^{-1}	3×10^{0}	Hepatotoxicity, hepatotoxicity
Trichloroethene**	6 x 10-3	ND	Kidney and liver effects; NA

* All toxicity values were taken from the 1991 Annual Volume of the Health Effects Assessment Summary Tables, except for the Chronic Reference Dose for Tetrachloroethene. That value was taken from the Integrated Risk Information System Database.
**Toxicity Information provided by the Environmental Criteria and Assessment Office (Appendix III).
ND - Not Determined
NA - Not Applicable

predicts blood lead levels for children. Predicted blood lead levels greater than 10 ug/dL are considered to present a health hazard. The model was run using a groundwater concentration of 0.000918 mg/L with default values for soil, air, food and paint.

4.2 Carcinogenic Effects

Slope factors found in IRIS and HEAST are used to assess carcinogenic effects for specific contaminants. A Slope factor is a plausible upper-bound estimate of the probability of a response per unit intake of a chemical expressed over a lifetime. Slope factors for the specific contaminants, weight of evidence classifications for carcinogenicity, and site of tumor data are summarized in Table 7.

5.0 RISK CHARACTERIZATION

5.1 Noncarcinogenic Risk

Noncancer hazard quotients are calculated for each contaminant in each pathway by dividing the Chronic Daily Intake (CDI) by the RfD (or RfC). The noncancer hazard quotients within an exposure pathway are summed to give the pathway hazard index. The Total Hazard Index is then calculated by summing the pathway hazard indices. According to RAGS (EPA 1989), adverse, noncarcinogenic effects in exposed human populations (including any sensitive individuals) are unlikely to occur when hazard indices are less than one (1.0).

5.1.1 RME 1

The pathway hazard indices for ingestion of contaminated drinking water and inhalation of volatilized VOCs during showering were 0.00031 and 0.000024, respectively. Chemicals which drove the risk assessment were 1,1 dichloroethene for ingestion of contaminated drinking water and 1,1 dichloroethane for inhalation of volatilized VOCs. The Total Hazard Index calculated for RME 1 was 0.00033 (Table 8). Because this is less than 1.0, potential health risks are not indicated for a child living across the Blue River from the North End Site, ingesting 1.0 liter of contaminated drinking water per day and inhaling volatilized VOCs 0.25 hours per day, 365 days per year for 6 years.

The Lead Biokinetic Uptake Model was used to predict blood lead levels of a child living in the trailer park across from the North End Site. Groundwater concentrations of 0.000918 mg/L were used in the intake calculations. Blood lead levels between 2.77 and 3.21 ug/dL were predicted by the model. Because these values are well below 10 ug/dL,

Table 7.

Carcinogenic Toxicity Values for Chemicals of Concern Found at the North End Site, Kansas City, MO.*

Contaminant	Oral Slope Factor $(mg/kg/day)^{-1}$	Inhalation Slope Factor $(mg/kg/day)^{-1}$	Weight of Evidence Classification and Tumor Site (oral; inhalation)
Chloromethane	1.3×10^{-2}	6.0×10^{-3}	C - Kidney; kidney
1,1 Dichloroethane	ND	ND	C - NA; hemangiosarcoma
1,2 Dichloroethane	9.1×10^{-2}	9.1×10^{-2}	B2 - Circulatory; circulatory
1,1 Dichloroethene	6.0×10^{-1}	1.2×10^{0}	C - Kidney; adrenal
Trichloroethene	1.1×10^{-2}	1.7×10^{-2}	B2 - Lung; liver
Vinyl chloride	1.9×10^{0}	2.9×10^{-1}	A - Liver; lung

* All slope factors except for chloromethane and trichloroethene were obtained from the Integrated Risk Information System database. Slope Factors for chloromethane and trichloroethene were obtained from the 1991 Annual Volume of the Health Effects Assessment Summary Tables.
ND - Not Determined
NA - Not Applicable

Table 8.

Hazard Index Values for RME 1
North End Site, Kansas City, MO

Pathway: Ingestion of contaminated drinking water by a 15 kg child over a 6 year period.					
Chemical	**Concentration (mg/L)**	**Chronic Daily Intake (mg/kg/day)**	**RfD (mg/kg/day)**	**Hazard Index**	**Pathway Hazard Index**
1,2 Dichlorobenzene	0.0000521	3.2×10^{-6}	9×10^{-1}	3.6×10^{-6}	
1,1 Dichloroethane	0.00149	9.2×10^{-5}	1×10^{0}	9.2×10^{-5}	
1,1 Dichloroethene	0.00084	1.7×10^{-6}	9×10^{-3}	1.9×10^{-4}	
1,2 Dichloroethene	0.000058	1.2×10^{-7}	1×10^{-1}	1.2×10^{-6}	
Tetrachloroethene	0.0000855	1.7×10^{-7}	1×10^{-1}	1.7×10^{-6}	
1,1,1 Trichloroethane	0.000578	1.2×10^{-6}	9×10^{-1}	1.3×10^{-6}	
Trichloroethene	0.0000569	1.2×10^{-7}	6×10^{-3}	1.9×10^{-5}	**0.00031**
Pathway: Inhalation of volatized compounds during showering by a 15 kg child over a 6 year period.					
Chemical	**Concentration (mg/m^3)**	**Chronic Daily Intake (mg/kg/day)**	**RfC (mg/kg/day)**	**Hazard Index**	**Pathway Hazard Index**
Chloroethane	0.00039	5.0×10^{-6}	1×10^{1}	5.0×10^{-7}	
1,2 Dichlorobenzene	0.0000521	6.6×10^{-7}	4×10^{-1}	1.7×10^{-6}	
1,1 Dichloroethane	0.00149	1.9×10^{-5}	1×10^{0}	1.9×10^{-5}	
1,1,1 Trichloroethane	0.000578	7.4×10^{-6}	3×10^{0}	2.5×10^{-6}	**0.000024**
		Total Hazard Index			**0.00033**

predicted lead concentrations in groundwater are not expected to cause adverse health effects to a child living across the Blue River from the North End Site.

5.1.2 RME 2

The pathway hazard indices for ingestion of contaminated drinking water and inhalation of volatilized VOCs during showering were 0.004 and 0.000054, respectively. Chemicals which drove the risk assessment were 1,1 dichloroethene for ingestion of contaminated drinking water and 1,1 dichloroethane for inhalation of volatilized VOCs. The Total Hazard Index calculated for RME 2 was 0.004 (Table 9). Because this is less than 1.0, potential health risks are not indicated for an adult living across the Blue River from the North End Site, ingesting 2.0 liters of contaminated drinking water per day and inhaling volatilized VOCs 0.25 hours per day, 365 days per year for 30 years.

The Lead Biokinetic Uptake Model was used to predict blood lead levels of an adult living in the trailer park across from the North End Site. Groundwater concentrations of 0.000918 mg/L were used in the intake calculations. Blood lead levels between 2.77 and 3.21 ug/dL were predicted by the model. Because these values are well below 10 ug/dL, predicted lead concentrations in groundwater are not expected to cause adverse health effects to an adult living across the Blue River from the North End Site.

5.2 Carcinogenic Risk

Lifetime excess cancer risks are calculated for each contaminant in each pathway by multiplying the slope factor by the Chronic Daily Intake (CDI). Within a pathway, the chemical specific risks are summed to give the total pathway risk. The Total Lifetime Excess Cancer Risk is then determined by summing the total pathway risks. According to RAGS (EPA 1989), a cumulative site carcinogenic risk of 1 in 10,000 to 1 in 1,000,000 is considered protective of human health. Generally, remedial or removal actions are considered necessary when the cumulative carcinogenic risk exceeds this range (carcinogenic risk greater than 1 in 10,000).

5.2.1 RME 1

Pathway cancer risks for ingestion of contaminated drinking water and inhalation of volatilized VOCs during showering were 4 in 1,000,000 and 1 in 1,000,000, respectively. The chemical which drove the risk assessment was 1,1 dichloroethene for ingestion of contaminated drinking water and inhalation of volatilized VOCs. The Total Excess Lifetime Cancer Risk calculated for RME 1 was 5 in 1,000,000 (Table 10), which is well below the upper end of the acceptable range for a child living across the Blue River from the North

Table 9.

Hazard Index Values for RME 2
North End Site, Kansas City, MO.

Chemical	Concentration (mg/L)	Chronic Daily Intake (mg/kg/day)	RfD (mg/kg/day)	Hazard Index	Pathway Hazard Index
Pathway: Ingestion of contaminated drinking water by a 70 kg adult over a 30 year period.					
1,2 Dichlorobenzene	0.0000521	1.5×10^{-6}	9×10^{-2}	1.7×10^{-5}	
1,1 Dichloroethane	0.00149	4.2×10^{-5}	1×10^{-1}	4.2×10^{-4}	
1,1 Dichloroethene	0.00084	2.4×10^{-5}	9×10^{-3}	2.7×10^{-3}	
1,2 Dichloroethene	0.000058	1.6×10^{-6}	1×10^{-2}	1.6×10^{-4}	
Tetrachloroethene	0.0000855	2.4×10^{-6}	1×10^{-2}	2.4×10^{-4}	
1,1,1 Trichloroethane	0.000578	1.6×10^{-5}	9×10^{-2}	1.8×10^{-4}	
Trichloroethene	0.0000569	1.6×10^{-6}	6×10^{-3}	2.7×10^{-4}	**0.004**
Pathway: Inhalation of volatized compounds while showering by a 70 kg adult over a 30 year period.					
Chemical	**Concentration (mg/m^3)**	**Chronic Daily Intake (mg/kg/day)**	**RfC (mg/kg/day)**	**Hazard Index**	**Pathway Hazard Index**
Chloroethane	0.00039	1.2×10^{-6}	$1 \times 10\ 1$	1.2×10^{-7}	
1,2 Dichlorobenzene	0.0000521	1.5×10^{-7}	4×10^{-2}	3.8×10^{-6}	
1,1 Dichloroethane	0.00149	4.4×10^{-6}	1×10^{-1}	4.4×10^{-5}	
1,1,1 Trichloroethane	0.000578	1.7×10^{-6}	3×10^{-1}	5.7×10^{-6}	**0.000054**
		Total Hazard Index			**0.004**

Table 10.

Excess Lifetime Cancer Risks for RME 1
North End Site, Kansas City, MO.

Pathway: Ingestion of contaminated drinking water by a 15 kg child over a 6 year period.					
Chemical	**Concentration (mg/L)**	**Chronic Daily Intake (mg/kg/day)**	**Slope Factor (mg/kg/day)-1**	**Cancer Risk**	**Pathway Cancer Risk**
Chloromethane	0.0000972	5.5×10^{-7}	1.3×10^{-2}	7.2×10^{-9}	
1,2 Dichloroethane	0.0000481	2.7×10^{-7}	9.1×10^{-2}	2.5×10^{-8}	
1,1 Dichloroethene	0.00084	4.8×10^{-6}	6.0×10^{-1}	2.9×10^{-6}	
Trichloroethene	0.0000569	3.2×10^{-7}	1.1×10^{-2}	3.6×10^{-9}	
Vinyl Chloride	0.000116	6.7×10^{-7}	1.9×10^{0}	1.2×10^{-6}	**4.2×10^{-6}**
Pathway: Inhalation volatized compounds during showering by a 15 kg child over a 6 year period.					
Chemical	**Concentration (mg/m^3)**	**Chronic Daily Intake (mg/kg/day)**	**Slope Factor $(mg/kg/day)^{-1}$**	**Cancer Risk**	**Pathway Cancer Risk**
Chloromethane	0.0000972	1.2×10^{-7}	6.3×10^{-3}	7.6×10^{-10}	
1,2 Dichloroethane	0.0000481	5.7×10^{-8}	9.1×10^{-2}	5.1×10^{-9}	
1,1 Dichloroethene	0.00084	1.0×10^{-6}	1.2×10^{0}	1.2×10^{-6}	
Trichloroethene	0.0000569	6.7×10^{-8}	1.7×10^{-2}	1.0×10^{-9}	
Vinyl Chloride	0.000116	1.4×10^{-7}	2.9×10^{-1}	4.0×10^{-8}	**1.2×10^{-6}**
		Excess Lifetime Cancer Risk			**5.4×10^{-6}**

End Site, ingesting 1.0 liter of contaminated drinking water per day and inhaling volatilized VOCs 0.25 hours per day, 365 days per year for 6 years.

5.2.2 RME 2

Pathway cancer risks for ingestion of contaminated drinking water and inhalation of volatilized VOCs during showering were 9 in 1,000,000 and 3 in 100,000, respectively. The chemical which drove the risk assessment was 1,1 dichloroethene for ingestion of contaminated drinking water and inhalation of volatilized VOCs. The Total Excess Lifetime Cancer Risk calculated for RME 2 was 4 in 100,000 (Table 11), which is well below the upper end of the acceptable range for an adult living across the Blue River from the North End Site, ingesting 2.0 liters of contaminated drinking water per day and inhaling volatilized VOCs 0.25 hours per day, 365 days per year for 30 years.

5.3 Uncertainties

Several areas of uncertainty are inherent in the risk assessment process. Most intake variables used are 95% upper confidence limits of the mean variable value. This may overestimate the true risk posed by the site. Many RfDs, RfCs and SFs are based on toxicity tests carried out on animals. It is not known if results of these tests are applicable to humans (see discussion of Class C carcinogen slope factors at end of Uncertainties section).

The Lead Biokinetic Uptake Model used to predict blood lead levels was developed for children aged 0-6 years, the ages at which effects from lead exposure are most dramatic. Because the effects of lead exposure are less prominent in older children and adults, the risk for adults from ingesting lead at the North End Site is probably lower than estimated in this assessment.

Four contaminants of concern undetected during the second round of sampling were retained in this risk assessment based upon an assumption that mixing will occur during movement of groundwater. This assumption may over- or underestimate the true risk posed by the site.

A classification system has been developed by EPA to characterize the extent to which a compound is a <u>human</u> carcinogen. Evidence for the carcinogenicity of compounds is based upon the extent to which the compound has been shown to be carcinogenic to humans, animals or both. The compound is then given a provisional weight-of-evidence classification. EPA adjusts this provisional classification upward or downward based on

Table 11.

Excess Lifetime Cancer Risks for RME 2
North End Site, Kansas City, MO.

Pathway: Ingestion of contaminated drinking water by a 70 kg adult over a 30 year period.					
Chemical	**Concentration (mg/L)**	**Chronic Daily Intake (mg/kg/day)**	**Slope Factor $(mg/kg/day)^{-1}$**	**Cancer Risk**	**Pathway Cancer Risk**
Chloromethane	0.0000972	1.1×10^{-6}	1.3×10^{-2}	1.5×10^{-8}	
1,2 Dichloroethane	0.0000481	5.9×10^{-7}	9.1×10^{-2}	5.4×10^{-8}	
1,1 Dichloroethene	0.00084	1.0×10^{-5}	6.0×10^{-1}	6.2×10^{-6}	
Trichloroethene	0.0000569	7.0×10^{-7}	1.1×10^{-2}	7.7×10^{-9}	
Vinyl Chloride	0.000116	1.4×10^{-6}	1.9×10^{0}	2.7×10^{-6}	**9.0×10^{-6}**
Pathway: Inhalation of volatized compounds by a 70 kg adult while showering over a 30 year period.					
Chemical	**Concentration (mg/m^3)**	**Chronic Daily Intake (mg/kg/day)**	**Slope Factor $(mg/kg/day)^{-1}$**	**Cancer Risk**	**Pathway Cancer Risk**
Chloromethane	0.0000972	3.0×10^{-6}	6.3×10^{-3}	1.8×10^{-8}	
1,2 Dichloroethane	0.0000481	1.5×10^{-6}	9.1×10^{-2}	1.4×10^{-7}	
1,1 Dichloroethene	0.00084	2.6×10^{-5}	1.2×10^{0}	3.1×10^{-5}	
Trichloroethene	0.0000569	1.7×10^{-6}	1.7×10^{-2}	2.8×10^{-8}	
Vinyl Chloride	0.000116	3.6×10^{-6}	2.9×10^{-1}	1.0×10^{-6}	**3.2×10^{-5}**
		Excess Lifetime Cancer Risk			**4.1×10^{-5}**

other supporting evidence of carcinogenicity. EPA has identified six weight-of-evidence classes of carcinogens:

Class	Description
A	Human carcinogen
B1,B2	Probable human carcinogen -B1: Limited data for human carcinogenicity -B2: Inadequate or no evidence for human carcinogenicity
C	Possible human carcinogen
D	Not classifiable as to human carcinogenicity
E	Evidence of noncarcinogenicity for humans

Although slope factors for carcinogenic risk are provided in IRIS for Class A, B1, B2, and C carcinogens, the confidence that a compound is carcinogenic is much greater for Class A carcinogens than for Class B1 carcinogens, Class B1 than for Class B2, and so on. Slope factors are provided for Class C carcinogens in IRIS although a higher degree of uncertainty is attached to these values.

Although additional response actions do not appear to be necessary for the North End Site based upon either noncarcinogenic or carcinogenic risk, EPA points out that a substantial portion of the carcinogenic risk for both RME 1 and RME 2 comes from Class C carcinogens: chloromethane and 1,1-dichloroethene. Carcinogenic risk may therefore be somewhat less than that indicated if EPA later determines these Class C carcinogens are not carcinogenic or are less carcinogenic than the current slope factors provided in IRIS.

6.0 SUMMARY

The North End Site was used as a landfill for the burial of lead-contaminated wastes. A soil removal action was completed and groundwater monitoring wells were installed and sampled. Total lead and some VOCs were detected in groundwater under the site. EPA directed DOH to assess the risks posed to a hypothetical future off-site resident ingesting contaminated drinking water and inhaling volatilized VOCs while showering.

Two RMEs were considered for future land use: a child (RME 1) and an adult (RME 2) ingesting contaminated drinking water and inhaling volatilized VOCs while showering. Since the hazard indices were less than 1.0, EPA's risk assessment indicates that noncarcinogenic effects would be unlikely to occur for either RME 1(child) or RME 2 (adult). Pathway cancer risks ranged from 1 in 1,000,000 to 3 in 100,000, which are below the upper end of the acceptable range (1 in 10,000).

The Lead Biokinetic Uptake model predicted Blood Lead Levels ranging from 2.77 to 3.21 ug/dL. Because these levels do not exceed 10 ug/dL, a health hazard is not considered to exist from ingestion of contaminated drinking water across the Blue River from the North End Site.

LITERATURE CITED

Centers for Disease Control (CDC). 1991. Preventing Lead Poisoning in Children. U. S. Department of Health and Human Services, Atlanta, GA.

Environmental Criteria and Assessment Office (ECAO). 1991. Inhalation Exposure to Volatile Organic Compounds from Showering with Contaminated Groundwater (Park Mobile Home Court Site/Norfolk, NE).

Environmental Protection Agency (EPA). 1989. Risk assessment Guidance for Superfund. Volume I: Human Health Evaluation Manual (Part A). Office of Emergency and Remedial Response. EPA/540/1-89/002.

Environmental Protection Agency (EPA). 1990. Exposure Factors Handbook. Office of Health and Environmental Assessment. EPA/600/8-89/043.

Environmental Protection Agency (EPA). 1991a. Human Health Evaluation Manual, Supplemental Guidance: (Standard Default Exposure Factors.) OSWER Directive 9285.6-03.

Environmental Protection Agency (EPA). 1991b. Health Effects Assessment Summary Tables, Annual FY-1991. Office of Emergency and Remedial Response. OERR 9200.6-303 (91-1).

Environmental Protection Agency (EPA). 1991c. Risk Assessment Guidance for Superfund. Volume I: Human Health Evaluation Manual (Part B, Development of Risk-based Preliminary Remediation Goals). Office of Emergency and Remedial Response. OERR 9285.7-01B. December, 1991.

APPENDIX I
Summary of Groundwater Sampling Results
from the North End Site

TABLE 4

RESULTS OF GROUND WATER ANALYSES
MARCH 13, 1991

PARAMETER	UNITS	RAU-MW -001-15	RAU-MW -001D-15	RAU-MW -002-15	RAU-MW -003-15	RAU-MW -003D-15
WATER LEVEL	ft-msl	733.85	736.66	733.16	733.86	736.60
GENERAL CHEMISTRY						
pH	s.u.	6.4	7.1	6.1	6.5	7.0
SPECIFIC CONDUCTANCE	µmho/cm	1500	740	3100	850	540
ACIDITY AS $CaCO_3$	mg/l	<1.0	16	1.5	<1.0	<1.0
ALKALINITY AS $CaCO_3$	mg/l	470	430	240	300	280
NITRATES AS NO_3-N	mg/l	0.29	0.11	0.24	0.30	0.13
PHENOLICS	mg/l	<0.005	<0.005	0.009	0.008	0.006
TOTAL LEAD	mg/l	0.058	0.024	0.049	0.063	0.010
DISSOLVED LEAD	mg/l	<0.003	<0.003	<0.003	<0.003	<0.003
POLYCHLORINATED BIPHENYL	µg/l	<1.0	<1.0	<1.0	<1.0	<1.0
VOLATILE ORGANIC COMPOUNDS						
ACROLEIN	µg/l	<50	<50	<50	<125	<50
ACRYLONITRILE	µg/l	<50	<50	<50	<125	<50
BENZENE	µg/l	<5	<5	<5	<12	<5
BROMODICHLOROMETHANE	µg/l	<5	<5	<5	<12	<5
BROMOFORM	µg/l	<5	<5	<5	<12	<5
BROMOMETHANE	µg/l	<10	<10	<10	<25	<10
CARBON TETRACHLORIDE	µg/l	<5	<5	<5	<12	<5
CHLOROBENZENE	µg/l	<5	<5	<5	<12	<5
CHLOROETHANE	µg/l	160	<10	<10	<25	<10
2-CHLOROETHYLVINYL ETHER	µg/l	<10	<10	<10	<25	<10
CHLOROFORM	µg/l	<5	<5	<5	<12	<5
CHLOROMETHANE	µg/l	<10	<10	<10	<25	<10
DIBROMOCHLOROMETHANE	µg/l	<5	<5	<5	<12	<5
1,2-DICHLOROBENZENE	µg/l	<5	<5	<5	<12	<5
1,3-DICHLOROBENZENE	µg/l	<5	<5	<5	<12	<5
1,4-DICHLOROBENZENE	µg/l	<5	<5	<5	<12	<5
1,1-DICHLOROETHANE	µg/l	200	<5	8	35	<5
1,2-DICHLOROETHANE	µg/l	<5	<5	<5	<12	<5
1,1-DICHLOROETHENE	µg/l	29	<5	<5	270	<5
1,2-DICHLOROETHENE	µg/l	2	<5	<5	6	<5
1,2-DICHLOROPROPANE	µg/l	<5	<5	<5	<12	<5
cis-1,3-DICHLOROPROPANE	µg/l	<5	<5	<5	<12	<5
trans-1,3-DICHLOROPROPANE	µg/l	<5	<5	<5	<12	<5
ETHYLBENZENE	µg/l	<5	<5	<5	<12	<5
METHYLENE CHLORIDE	µg/l	<10	<10	<10	<25	<10
1,1,2,2-TETRACHLOROETHANE	µg/l	<5	<5	<5	<12	<5
TETRACHLOROETHENE	µg/l	<5	<5	<5	6	<5
TOLUENE	µg/l	<5	<5	<5	<12	<5
1,1,1-TRICHLOROETHANE	µg/l	38	<5	<5	67	<5
1,1,2-TRICHLOROETHANE	µg/l	<5	<5	<5	<12	<5
TRICHLOROETHENE	µg/l	<5	<5	<5	<12	<5
TRICHLOROFLUOROMETHANE	µg/l	<5	<5	<5	<12	<5
VINYL CHLORIDE	µg/l	23	<10	<10	16	<10
TOTAL VOCs	µg/l	452	0	8	400	0

TABLE 4
(CONTINUED)

PARAMETER	UNITS	RAU-MW -003A-15	RAU-MW -004-15	RAU-MW -005-15	RAU-MW -006-15	RAU-MW 006R-15
WATER LEVEL	ft-msl	734.69	739.36	743.69	743.91	REPLICATE
GENERAL CHEMISTRY						
pH	s.u.	6.2	6.6	6.0	6.3	6.4
SPECIFIC CONDUCTANCE	µmho/cm	600	2900	1600	960	950
ACIDITY AS $CaCO_3$	mg/l	<1.0	<1.0	<1.0	<1.0	<1.0
ALKALINITY AS $CaCO_3$	mg/l	140	450	110	140	140
NITRATES AS NO_3-N	mg/l	0.16	1.00	0.18	0.19	0.21
PHENOLICS	mg/l	0.007	0.006	0.006	<0.005	<0.005
TOTAL LEAD	mg/l	0.011	0.058	0.026	0.015	0.028
DISSOLVED LEAD	mg/l	<0.003	0.020	<0.003	<0.003	<0.003
POLYCHLORINATED BIPHENYL	µg/l	<1.0	<1.0	<1.0	<1.0	<1.0
VOLATILE ORGANIC COMPOUNDS						
ACROLEIN	µg/l	<50	<50	<50	<50	<50
ACRYLONITRILE	µg/l	<50	<50	<50	<50	<50
BENZENE	µg/l	<5	<5	<5	<5	<5
BROMODICHLOROMETHANE	µg/l	<5	<5	<5	<5	<5
BROMOFORM	µg/l	<5	<5	<5	<5	<5
BROMOMETHANE	µg/l	<10	<10	<10	<10	<10
CARBON TETRACHLORIDE	µg/l	<5	<5	<5	<5	<5
CHLOROBENZENE	µg/l	<5	<5	<5	<5	<5
CHLOROETHANE	µg/l	<10	<10	<10	<10	<10
2-CHLOROETHYLVINYL ETHER	µg/l	<10	<10	<10	<10	<10
CHLOROFORM	µg/l	<5	<5	<5	<5	<5
CHLOROMETHANE	µg/l	20	<10	<10	<10	<10
DIBROMOCHLOROMETHANE	µg/l	<5	<5	<5	<5	<5
1,2-DICHLOROBENZENE	µg/l	<5	<5	<5	<5	<5
1,3-DICHLOROBENZENE	µg/l	<5	<5	<5	<5	<5
1,4-DICHLOROBENZENE	µg/l	<5	<5	<5	<5	<5
1,1-DICHLOROETHANE	µg/l	9	6	<5	<5	<5
1,2-DICHLOROETHANE	µg/l	<5	<5	<5	<5	<5
1,1-DICHLOROETHENE	µg/l	120	29	<5	<5	<5
1,2-DICHLOROETHENE	µg/l	<5	<5	<5	<5	<5
1,2-DICHLOROPROPANE	µg/l	<5	<5	<5	<5	<5
cis-1,3-DICHLOROPROPANE	µg/l	<5	<5	<5	<5	<5
trans-1,3-DICHLOROPROPANE	µg/l	<5	<5	<5	<5	<5
ETHYLBENZENE	µg/l	<5	<5	<5	<5	<5
METHYLENE CHLORIDE	µg/l	<10	<10	<10	<10	<10
1,1,2,2-TETRACHLOROETHANE	µg/l	<5	<5	<5	<5	<5
TETRACHLOROETHENE	µg/l	28	<5	<5	<5	<5
TOLUENE	µg/l	<5	<5	<5	<5	<5
1,1,1-TRICHLOROETHANE	µg/l	37	17	<5	<5	<5
1,1,2-TRICHLOROETHANE	µg/l	<5	<5	<5	<5	<5
TRICHLOROETHENE	µg/l	3	<5	<5	<5	<5
TRICHLOROFLUOROMETHANE	µg/l	<5	<5	<5	<5	<5
VINYL CHLORIDE	µg/l	<10	<10	<10	<10	<10
TOTAL VOCs	µg/l	217	52	0	0	0

TABLE 4
(CONTINUED)

PARAMETER	UNITS	RAU-MW -007-15	RAU-MW -008-15	RAU-MW -009-15	RAU-MW -010-15	RAU-MW -011-15
WATER LEVEL	ft-msl	746.43	738.31	743.91	732.83	733.08
GENERAL CHEMISTRY						
pH	s.u.	6.3	6.9	6.2	5.9	6.6
SPECIFIC CONDUCTANCE	µmho/cm	910	940	510	2900	960
ACIDITY AS CaCO3	mg/l	<1.0	<1.0	<1.0	8	<1.0
ALKALINITY AS CaCO3	mg/l	83	460	68	170	200
NITRATES AS NO3-N	mg/l	1.30	0.17	<0.10	0.44	0.26
PHENOLICS	mg/l	<0.005	<0.005	<0.005	<0.005	<0.005
TOTAL LEAD	mg/l	0.009	0.083	0.015	0.066	0.030
DISSOLVED LEAD	mg/l	<0.003	<0.003	<0.003	<0.003	<0.003
POLYCHLORINATED BIPHENYL	µg/l	<1.0	<1.0	<1.0	<1.0	<1.0
VOLATILE ORGANIC COMPOUNDS						
ACROLEIN	µg/l	<50	<50	<50	<250	<50
ACRYLONITRILE	µg/l	<50	<50	<50	<250	<50
BENZENE	µg/l	<5	<5	<5	<25	<5
BROMODICHLOROMETHANE	µg/l	<5	<5	<5	<25	<5
BROMOFORM	µg/l	<5	<5	<5	<25	<5
BROMOMETHANE	µg/l	<10	<10	<10	<50	<10
CARBON TETRACHLORIDE	µg/l	<5	<5	<5	<25	<5
CHLOROBENZENE	µg/l	<5	<5	<5	<25	<5
CHLOROETHANE	µg/l	<10	<10	<10	37	<10
2-CHLOROETHYLVINYL ETHER	µg/l	<10	<10	<10	<50	<10
CHLOROFORM	µg/l	<5	<5	<5	<25	<5
CHLOROMETHANE	µg/l	<10	<10	<10	<50	<10
DIBROMOCHLOROMETHANE	µg/l	<5	<5	<5	<25	<5
1,2-DICHLOROBENZENE	µg/l	<5	<5	15	<25	<5
1,3-DICHLOROBENZENE	µg/l	<5	<5	<5	<25	<5
1,4-DICHLOROBENZENE	µg/l	<5	<5	<5	<25	<5
1,1-DICHLOROETHANE	µg/l	<5	<5	<5	580	8
1,2-DICHLOROETHANE	µg/l	<5	<5	<5	<25	<5
1,1-DICHLOROETHENE	µg/l	<5	<5	<5	83	<5
1,2-DICHLOROETHENE	µg/l	<5	<5	3	23	<5
1,2-DICHLOROPROPANE	µg/l	<5	<5	<5	<25	<5
cis-1,3-DICHLOROPROPANE	µg/l	<5	<5	<5	<25	<5
trans-1,3-DICHLOROPROPANE	µg/l	<5	<5	<5	<25	<5
ETHYLBENZENE	µg/l	<5	<5	<5	<25	<5
METHYLENE CHLORIDE	µg/l	<10	<10	<10	<50	<10
1,1,2,2-TETRACHLOROETHANE	µg/l	<5	<5	<5	<25	<5
TETRACHLOROETHENE	µg/l	<5	<5	<5	<25	<5
TOLUENE	µg/l	<5	<5	<5	<25	<5
1,1,1-TRICHLOROETHANE	µg/l	<5	<5	<5	210	<5
1,1,2-TRICHLOROETHANE	µg/l	<5	<5	<5	<25	<5
TRICHLOROETHENE	µg/l	<5	<5	13	<25	<5
TRICHLOROFLUOROMETHANE	µg/l	<5	<5	<5	<25	<5
VINYL CHLORIDE	µg/l	<10	<10	<10	34	<10
TOTAL VOCs	µg/l	0	0	31	967	8

TABLE 4
(CONTINUED)

PARAMETER	UNITS	RAU-MW -012-15	RAU-MW -012R-15	RAU-MW -000-15	TRIP BLANK
WATER LEVEL	ft-msl	733.47	REPLICATE	BLANK	
GENERAL CHEMISTRY					
pH	s.u.	6.6		6.6	
SPECIFIC CONDUCTANCE	µmho/cm	1600		100	
ACIDITY AS CaCO3	mg/l	<1.0		11.	
ALKALINITY AS CaCO3	mg/l	680		7.8	
NITRATES AS NO3-N	mg/l	0.43		0.16	
PHENOLICS	mg/l	<0.005		<0.005	
TOTAL LEAD	mg/l	0.100		<0.003	
DISSOLVED LEAD	mg/l	<0.003		<0.003	
POLYCHLORINATED BIPHENYL	µg/l	<1.0		<1.0	
VOLATILE ORGANIC COMPOUNDS					
ACROLEIN	µg/l	<50	<50		<50
ACRYLONITRILE	µg/l	<50	<50		<50
BENZENE	µg/l	<5	<5		<5
BROMODICHLOROMETHANE	µg/l	<5	<5		<5
BROMOFORM	µg/l	<5	<5		<5
BROMOMETHANE	µg/l	<10	<10		<10
CARBON TETRACHLORIDE	µg/l	<5	<5		<5
CHLOROBENZENE	µg/l	<5	<5		<5
CHLOROETHANE	µg/l	<10	<10		<10
2-CHLOROETHYLVINYL ETHER	µg/l	<10	<10		<10
CHLOROFORM	µg/l	<5	<5		<5
CHLOROMETHANE	µg/l	<10	<10		<10
DIBROMOCHLOROMETHANE	µg/l	<5	<5		<5
1,2-DICHLOROBENZENE	µg/l	<5	<5		<5
1,3-DICHLOROBENZENE	µg/l	<5	<5		<5
1,4-DICHLOROBENZENE	µg/l	<5	<5		<5
1,1-DICHLOROETHANE	µg/l	120	120		<5
1,2-DICHLOROETHANE	µg/l	<5	<5		<5
1,1-DICHLOROETHENE	µg/l	<5	<5		<5
1,2-DICHLOROETHENE	µg/l	9	9		<5
1,2-DICHLOROPROPANE	µg/l	<5	<5		<5
cis-1,3-DICHLOROPROPANE	µg/l	<5	<5		<5
trans-1,3-DICHLOROPROPANE	µg/l	<5	<5		<5
ETHYLBENZENE	µg/l	<5	<5		<5
METHYLENE CHLORIDE	µg/l	<10	<10		<10
1,1,2,2-TETRACHLOROETHANE	µg/l	<5	<5		<5
TETRACHLOROETHENE	µg/l	<5	<5		<5
TOLUENE	µg/l	<5	<5		<5
1,1,1-TRICHLOROETHANE	µg/l	<5	<5		<5
1,1,2-TRICHLOROETHANE	µg/l	<5	<5		<5
TRICHLOROETHENE	µg/l	<5	<5		<5
TRICHLOROFLOUROMETHANE	µg/l	<5	<5		<5
VINYL CHLORIDE	µg/l	8	10		<10
TOTAL VOCs	µg/l	137	139		0

TABLE 5

RESULTS OF GROUND WATER ANALYSES
MAY 3,1991

PARAMETER	UNITS	RAU-MW -001-16	RAU-MW -001D-16	RAU-MW -002A-16	RAU-MW -003-16	RAU-MW -003D-16
WATER LEVEL	ft-msl	734.68	737.61		734.92	737.21
GENERAL CHEMISTRY						
pH	s.u.	6.4	6.8	6.0	6.6	6.9
SPECIFIC CONDUCTANCE	µmho/cm	1500	700	2900	780	580
ALKALINITY AS $CaCO_3$	mg/l	460	410	290	330	330
NITRATES AS NO_3-N	mg/l	0.32	0.20	0.40	0.40	0.14
PHENOLICS	mg/l	<0.006	<0.006	<0.006	<0.006	<0.006
TOTAL LEAD	mg/l	0.130	0.029	0.120	0.130	0.130
DISSOLVED LEAD	mg/l	<0.003	<0.003	<0.003	<0.003	<0.003
POLYCHLORINATED BIPHENYLS	µg/l	<1.0	<1.0	<1.0	<1.0	<1.0
VOLATILE ORGANIC COMPOUNDS						
ACROLEIN	µg/l	<50	<50	<50	<50	<50
ACRYLONITRILE	µg/l	<50	<50	<50	<50	<50
BENZENE	µg/l	<5	<5	<5	<5	<5
BROMODICHLOROMETHANE	µg/l	<5	<5	<5	<5	<5
BROMOFORM	µg/l	<5	<5	<5	<5	<5
BROMOMETHANE	µg/l	<10	<10	<10	<10	<10
CARBON TETRACHLORIDE	µg/l	<5	<5	<5	<5	<5
CHLOROBENZENE	µg/l	<5	<5	<5	<5	<5
CHLOROETHANE	µg/l	180	<10	<10	<10	<10
2-CHLOROETHYLVINYL ETHER	µg/l	<10	<10	<10	<10	<10
CHLOROFORM	µg/l	<5	<5	<5	<5	<5
CHLOROMETHANE	µg/l	<10	<10	<10	<10	<10
DIBROMOCHLOROMETHANE	µg/l	<5	<5	<5	<5	<5
1,2-DICHLOROBENZENE	µg/l	<5	<5	<5	<5	<5
1,3-DICHLOROBENZENE	µg/l	<5	<5	<5	<5	<5
1,4-DICHLOROBENZENE	µg/l	<5	<5	<5	<5	<5
1,1-DICHLOROETHANE	µg/l	317	<5	<5	44	<5
1,2-DICHLOROETHANE	µg/l	<5	<5	<5	<5	<5
1,1-DICHLOROETHENE	µg/l	86	<5	4.8(J)	410	<5
1,2-DICHLOROETHENE	µg/l	<5	<5	<5	<5	<5
1,2-DICHLOROPROPANE	µg/l	<5	<5	<5	<5	<5
cis-1,3-DICHLOROPROPANE	µg/l	<5	<5	<5	<5	<5
trans-1,3-DICHLOROPROPANE	µg/l	<5	<5	<5	<5	<5
ETHYLBENZENE	µg/l	<5	<5	<5	<5	<5
METHYLENE CHLORIDE	µg/l	<10	<10	<10	<10	<10
1,1,2,2-TETRACHLOROETHANE	µg/l	<5	<5	<5	<5	<5
TETRACHLOROETHENE	µg/l	<5	<5	<5	<5	<5
TOLUENE	µg/l	<5	<5	<5	<5	<5
1,1,1-TRICHLOROETHANE	µg/l	85	<5	<5	98	<5
1,1,2-TRICHLOROETHANE	µg/l	<5	<5	<5	<5	<5
TRICHLOROETHENE	µg/l	<5	<5	<5	<5	<5
TRICHLOROFLOUROMETHANE	µg/l	<5	<5	<5	<5	<5
VINYL CHLORIDE	µg/l	<10	<10	<10	<10	<10
TOTAL VOCs	µg/l	668	0	5	552	0

TABLE 5
(CONTINUED)

PARAMETER	UNITS	RAU-MW -003A-16	RAU-MW -004-16	RAU-MW -005-16	RAU-MW -006-16	RAU-MW 006R-16
WATER LEVEL	ft-msl	736.38	738.91	743.64	744.65	REPLICATE
GENERAL CHEMISTRY						
pH	s.u.	6.1	6.3	5.9	6.2	6.1
SPECIFIC CONDUCTANCE	µmho/cm	500	2800	1600	970	980
ALKALINITY AS $CaCO_3$	mg/l	120	440	100	110	120
NITRATES AS NO_3-N	mg/l	0.78	0.26	0.16	0.20	0.44
PHENOLICS	mg/l	<0.006	0.008		0.006	<0.005
TOTAL LEAD	mg/l	0.014	0.400	0.037	0.040	0.086
DISSOLVED LEAD	mg/l	<0.003	0.013	<0.003	<0.003	<0.003
POLYCHLORINATED BIPHENYLS	µg/l	<1.0	<1.0	<1.0	<1.0	<1.0
VOLATILE ORGANIC COMPOUNDS						
ACROLEIN	µg/l	<50	<50	<50	<50	<50
ACRYLONITRILE	µg/l	<50	<50	<50	<50	<50
BENZENE	µg/l	<5	<5	<5	<5	<5
BROMODICHLOROMETHANE	µg/l	<5	<5	<5	<5	<5
BROMOFORM	µg/l	<5	<5	<5	<5	<5
BROMOMETHANE	µg/l	<10	<10	<10	<10	<10
CARBON TETRACHLORIDE	µg/l	<5	<5	<5	<5	<5
CHLOROBENZENE	µg/l	<5	<5	<5	<5	<5
CHLOROETHANE	µg/l	<10	<10	<10	<10	<10
2-CHLOROETHYLVINYL ETHER	µg/l	<10	<10	<10	<10	<10
CHLOROFORM	µg/l	<5	<5	<5	<5	<5
CHLOROMETHANE	µg/l	<10	<10	<10	<10	<10
DIBROMOCHLOROMETHANE	µg/l	<5	<5	<5	<5	<5
1,2-DICHLOROBENZENE	µg/l	<5	<5	<5	<5	<5
1,3-DICHLOROBENZENE	µg/l	<5	<5	<5	<5	<5
1,4-DICHLOROBENZENE	µg/l	<5	<5	<5	<5	<5
1,1-DICHLOROETHANE	µg/l	<5	4.5(J)	<5	<5	<5
1,2-DICHLOROETHANE	µg/l	9.1	<5	<5	<5	<5
1,1-DICHLOROETHENE	µg/l	74	7.6	<5	<5	<5
1,2-DICHLOROETHENE	µg/l	<5	<5	<5	<5	<5
1,2-DICHLOROPROPANE	µg/l	<5	<5	<5	<5	<5
cis-1,3-DICHLOROPROPANE	µg/l	<5	<5	<5	<5	<5
trans-1,3-DICHLOROPROPANE	µg/l	<5	<5	<5	<5	<5
ETHYLBENZENE	µg/l	<5	<5	<5	<5	<5
METHYLENE CHLORIDE	µg/l	<10	<10	<10	<10	<10
1,1,2,2-TETRACHLOROETHANE	µg/l	<5	<5	<5	<5	<5
TETRACHLOROETHENE	µg/l	30	<5	<5	<5	<5
TOLUENE	µg/l	<5	<5	<5	<5	<5
1,1,1-TRICHLOROETHANE	µg/l	36	13	<5	<5	<5
1,1,2-TRICHLOROETHANE	µg/l	<5	<5	<5	<5	<5
TRICHLOROETHENE	µg/l	3(J)	<5	<5	<5	<5
TRICHLOROFLOUROMETHANE	µg/l	<5	<5	<5	<5	<5
VINYL CHLORIDE	µg/l	<10	<10	<10	<10	<10
TOTAL VOCs	µg/l	152.1	25.1	0	0	0

TABLE 5
(CONTINUED)

PARAMETER	UNITS	RAU-MW -007-16	RAU-MW -008-16	RAU-MW -009-16	RAU-MW -010-16	RAU-MW -011-16
WATER LEVEL	ft-msl	746.69	738.98	745.46	733.17	733.23
GENERAL CHEMISTRY						
pH	s.u.	6.1	6.8	6.0	5.9	6.8
SPECIFIC CONDUCTANCE	µmho/cm	920	960	520	25000	970
ALKALINITY AS CaCO3	mg/l	77	450	68	190	300
NITRATES AS NO3-N	mg/l	0.78	0.26	0.21	0.20	0.22
PHENOLICS	mg/l	0.007	0.006	<0.005	<0.006	<0.006
TOTAL LEAD	mg/l	0.010	0.033	0.013	0.054	0.009
DISSOLVED LEAD	mg/l	<0.003	<0.003	<0.003	<0.003	<0.003
POLYCHLORINATED BIPHENYLS	µg/l	<1.0	<1.0	<1.0	<1.0	<1.0
VOLATILE ORGANIC COMPOUNDS						
ACROLEIN	µg/l	<50	<50	<50	<250	<50
ACRYLONITRILE	µg/l	<50	<50	<50	<250	<50
BENZENE	µg/l	<5	<5	<5	<25	<5
BROMODICHLOROMETHANE	µg/l	<5	<5	<5	<25	<5
BROMOFORM	µg/l	<5	<5	<5	<25	<5
BROMOMETHANE	µg/l	<10	<10	<10	<50	<10
CARBON TETRACHLORIDE	µg/l	<5	<5	<5	<25	<5
CHLOROBENZENE	µg/l	<5	<5	<5	<25	<5
CHLOROETHANE	µg/l	<10	<10	<10	<50	<10
2-CHLOROETHYLVINYL ETHER	µg/l	<10	<10	<10	<50	<10
CHLOROFORM	µg/l	<5	<5	<5	<25	<5
CHLOROMETHANE	µg/l	<10	<10	<10	<50	<10
DIBROMOCHLOROMETHANE	µg/l	<5	<5	<5	<25	<5
1,2-DICHLOROBENZENE	µg/l	<5	<5	<5	<25	<5
1,3-DICHLOROBENZENE	µg/l	<5	<5	<5	<25	<5
1,4-DICHLOROBENZENE	µg/l	<5	<5	<5	<25	<5
1,1-DICHLOROETHANE	µg/l	<5	<5	<5	640	13
1,2-DICHLOROETHANE	µg/l	<5	<5	<5	<25	<5
1,1-DICHLOROETHENE	µg/l	<5	<5	<5	64	<5
1,2-DICHLOROETHENE	µg/l	<5	<5	<5	<25	<5
1,2-DICHLOROPROPANE	µg/l	<5	<5	<5	<25	<5
cis-1,3-DICHLOROPROPANE	µg/l	<5	<5	<5	<25	<5
trans-1,3-DICHLOROPROPANE	µg/l	<5	<5	<5	<25	<5
ETHYLBENZENE	µg/l	<5	<5	<5	<25	<5
METHYLENE CHLORIDE	µg/l	<10	<10	<10	<50	<10
1,1,2,2-TETRACHLOROETHANE	µg/l	<5	<5	<5	<25	<5
TETRACHLOROETHENE	µg/l	<5	<5	<5	<25	<5
TOLUENE	µg/l	<5	<5	<5	<25	<5
1,1,1-TRICHLOROETHANE	µg/l	<5	<5	<5	230	<5
1,1,2-TRICHLOROETHANE	µg/l	<5	<5	<5	<25	<5
TRICHLOROETHENE	µg/l	<5	<5	12	<25	<5
TRICHLOROFLOUROMETHANE	µg/l	<5	<5	<5	<25	<5
VINYL CHLORIDE	µg/l	<10	<10	<10	35 (J)	<10
TOTAL VOCs	µg/l	0	0	12	969	13

TABLE 5
(CONTINUED)

PARAMETER	UNITS	RAU-MW -012-16	RAU-MW -012R-16	RAU-MW -000-16	TRIP BLANK
WATER LEVEL	ft-msl	734.39	REPLICATE	BLANK	
GENERAL CHEMISTRY					
pH	s.u.	6.6		7.0	
SPECIFIC CONDUCTANCE	µmho/cm	1600		57	
ALKALINITY AS CaCO3	mg/l	740		15	
NITRATES AS NO3-N	mg/l	0.10		<0.10	
PHENOLICS	mg/l	<0.005		0.010	
TOTAL LEAD	mg/l	0.038		0.012	
DISSOLVED LEAD	mg/l	<0.003		<0.003	
POLYCHLORINATED BIPHENYLS	µg/l	<1.0		<1.0	
VOLATILE ORGANIC COMPOUNDS					
ACROLEIN	µg/l	<50	<50	<50	<50
ACRYLONITRILE	µg/l	<50	<50	<50	<50
BENZENE	µg/l	<5	<5	<5	<5
BROMODICHLOROMETHANE	µg/l	<5	<5	<5	<5
BROMOFORM	µg/l	<5	<5	<5	<5
BROMOMETHANE	µg/l	<10	<10	<10	<10
CARBON TETRACHLORIDE	µg/l	<5	<5	<5	<5
CHLOROBENZENE	µg/l	<5	<5	<5	<5
CHLOROETHANE	µg/l	<10	<10	<10	<10
2-CHLOROETHYLVINYL ETHER	µg/l	<10	<10	<10	<10
CHLOROFORM	µg/l	<5	<5	<5	<5
CHLOROMETHANE	µg/l	<10	<10	<10	<10
DIBROMOCHLOROMETHANE	µg/l	<5	<5	<5	<5
1,2-DICHLOROBENZENE	µg/l	<5	<5	<5	<5
1,3-DICHLOROBENZENE	µg/l	<5	<5	<5	<5
1,4-DICHLOROBENZENE	µg/l	<5	<5	<5	<5
1,1-DICHLOROETHANE	µg/l	37	33	<5	<5
1,2-DICHLOROETHANE	µg/l	<5	<5	<5	<5
1,1-DICHLOROETHENE	µg/l	<5	<5	<5	<5
1,2-DICHLOROETHENE	µg/l	<5	<5	<5	<5
1,2-DICHLOROPROPANE	µg/l	<5	<5	<5	<5
cis-1,3-DICHLOROPROPANE	µg/l	<5	<5	<5	<5
trans-1,3-DICHLOROPROPANE	µg/l	<5	<5	<5	<5
ETHYLBENZENE	µg/l	<5	<5	<5	<5
METHYLENE CHLORIDE	µg/l	<10	<10	<10	<10
1,1,2,2-TETRACHLOROETHANE	µg/l	<5	<5	<5	<5
TETRACHLOROETHENE	µg/l	<5	<5	<5	<5
TOLUENE	µg/l	<5	<5	<5	4 (J)
1,1,1-TRICHLOROETHANE	µg/l	<5	<5	<5	<5
1,1,2-TRICHLOROETHANE	µg/l	<5	<5	<5	<5
TRICHLOROETHENE	µg/l	<5	<5	<5	<5
TRICHLOROFLOUROMETHANE	µg/l	<5	<5	<5	<5
VINYL CHLORIDE	µg/l	<10	<10	<10	<10
TOTAL VOCs	µg/l	37	33	0	4

APPENDIX II

Risk Calculations

North End Site

Intake Calculations

Ingestion of Drinking Water:

Intake= ((IR)(EF)(ED))/((BW)(AT))

Variable Values	Adult	Child	
Ingestion Rate (IR)	2 L/day	1 L/day	
Exposure Frequency (EF)	365 days/year	365 days/year	
Exposure Duration (ED)	30 years	6 years	
Body Weight (BW)	70 kg	15 kg	
Averaging Time (AT)	10950 days	2190 days	(Noncarcinogenic Effects)
	25550 days	25550 days	(Carcinogenic Effects)

Inhalation of Volatized Chemicals:

Intake= ((IR)(ET)(EF)(ED))/((BW)(AT))

Variable Values	Adult	Child
Ingestion Rate (IR)	0.83 m3/hr	0.83 m3/hr
Exposure Time (ET)	0.25 hr/ day	0.25 hr/ day
Exposure Frequency (EF)	365 days/year	365 days/year
Exposure Duration (ED)	30 years	6 years
Body Weight (BW)	70 kg	15 kg
Averaging Time (AT)	10950 days	2190 days
	25550 days	25550 days

Final Intake = (Intake)(Chemical Concentration)

Hazard Index = (Final Intake)/(Reference Dose)

Cancer Risk = (Final Intake)(Slope Factor)

FINAL INTAKE CALCULATIONS -Adult, Noncancer

Pathway: Ingestion of contaminated drinking water

Chemical	Concentration	Intake	Final Intake	RfD	Hazard Index
Chloroethane	0.00039	0.028571429	1.11429E-05		ND
Chloromethane	0.0000972	0.028571429	2.77714E-06		ND
1,2 Dichlorobenzene	0.0000521	0.028571429	1.48857E-06	0.09	1.65397E-05
1,1 Dichloroethane	0.00149	0.028571429	4.25714E-05	0.1	0.000425714
1,2 Dichloroethane	0.0000481	0.028571429	1.37429E-06		ND
1,1 Dichloroethene	0.00084	0.028571429	0.000024	0.009	0.002666667
1,2 Dichloroethene	0.000058	0.028571429	1.65714E-06	0.01	0.000165714
Tetrachloroethene	0.0000855	0.028571429	2.44286E-06	0.01	0.000244286
1,1,1 Trichloroethane	0.000578	0.028571429	1.65143E-05	0.09	0.000183492
Trichloroethene	0.0000569	0.028571429	1.62571E-06	0.006	0.000271
Vinyl Chloride	0.000116	0.028571429	3.31429E-06		ND
			Pathway Hazard Index		**0.003973412**

Pathway: Inhalation of contaminated air

Chemical	Concentration	Intake	Final Intake	RfC	Hazard Index
Chloroethane	0.00039	0.002964286	1.15607E-06	10	1.16E-07
Chloromethane	0.0000972	0.002964286	2.88129E-07		ND
1,2 Dichlorobenzene	0.0000521	0.002964286	1.54439E-07	0.04	3.86E-06
1,1 Dichloroethane	0.00149	0.002964286	4.41679E-06	0.1	4.41679E-05
1,2 Dichloroethane	0.0000481	0.002964286	1.42582E-07		ND
1,1 Dichloroethene	0.00084	0.002964286	0.00000249		ND
1,2 Dichloroethene	0.000058	0.002964286	1.71929E-07		ND
Tetrachloroethene	0.0000855	0.002964286	2.53446E-07		ND
1,1,1 Trichloroethane	0.000578	0.002964286	1.71336E-06	0.3	5.70E-06
Trichloroethene	0.0000569	0.002964286	1.68668E-07		ND
Vinyl Chloride	0.000116	0.002964286	3.43857E-07		ND
			Pathway Hazard Index		**0.000053843**

FINAL INTAKE CALCULATIONS -Child, Noncancer

Pathway: Ingestion of contaminated drinking water

Chemical	Concentration	Intake	Final Intake	RfD	Hazard Index
Chloroethane	0.00039	0.061603376	2.40253E-05		ND
Chloromethane	0.0000972	0.061603376	5.98785E-06		ND
1,2 Dichlorobenzene	0.0000521	0.061603376	3.20954E-06	0.9	3.56615E-06
1,1 Dichloroethane	0.00149	0.061603376	9.1789E-05	1	9.1789E-05
1,2 Dichloroethane	0.0000481	0.061603376	2.96312E-06		ND
1,1 Dichloroethene	0.00084	0.002025316	1.70127E-06	0.009	0.00018903
1,2 Dichloroethene	0.000058	0.002025316	1.17468E-07	0.1	1.17468E-06
Tetrachloroethene	0.0000855	0.002025316	1.73165E-07	0.1	1.73165E-06
1,1,1 Trichloroethane	0.000578	0.002025316	1.17063E-06	0.09	1.30E-06
Trichloroethene	0.0000569	0.002025316	1.15241E-07	0.006	0.0000192
Vinyl Chloride	0.000116	0.002025316	2.34937E-07		ND
			Pathway Hazard Index		**0.000307991**

Pathway: Inhalation of contaminated air

Chemical	Concentration	Intake	Final Intake	RfC	Hazard Index
Chloroethane	0.00039	0.0127827	4.98525E-06	10	4.99E-07
Chloromethane	0.0000972	0.0127827	1.24248E-06		ND
1,2 Dichlorobenzene	0.0000521	0.0127827	6.65979E-07	0.4	1.66E-06
1,1 Dichloroethane	0.00149	0.0127827	1.90462E-05	1	1.90E-05
1,2 Dichloroethane	0.0000481	0.0127827	6.14848E-07		ND
1,1 Dichloroethene	0.00084	0.0127827	1.07375E-05		ND
1,2 Dichloroethene	0.000058	0.0127827	7.41397E-07		ND
Tetrachloroethene	0.0000855	0.0127827	1.09292E-06		ND
1,1,1 Trichloroethane	0.000578	0.0127827	7.3884E-06	3	2.46E-06
Trichloroethene	0.0000569	0.0127827	7.27336E-07		ND
Vinyl Chloride	0.000116	0.0127827	1.48279E-06		ND
			Pathway Hazard Index		**0.000023619**

FINAL INTAKE CALCULATIONS - Adult, Cancer

Pathway: Ingestion of contaminated drinking water

Chemical	Concentration	Intake	Final Intake	Slope Factor	Cancer Risk
Chloroethane	0.00039	0.012244898	4.77551E-06		ND
Chloromethane	0.0000972	0.012244898	1.1902E-06	0.013	1.54727E-08
1,2 Dichlorobenzene	0.0000521	0.012244898	6.37959E-07		ND
1,1 Dichloroethane	0.00149	0.012244898	1.82449E-05		ND
1,2 Dichloroethane	0.0000481	0.012244898	5.8898E-07	0.091	5.35971E-08
1,1 Dichloroethene	0.00084	0.012244898	1.02857E-05	0.6	6.17143E-06
1,2 Dichloroethene	0.000058	0.012244898	7.10204E-07		ND
Tetrachloroethene	0.0000855	0.012244898	1.04694E-06		ND
1,1,1 Trichloroethane	0.000578	0.012244898	7.07755E-06		ND
Trichloroethene	0.0000569	0.012244898	6.96735E-07	0.011	7.66408E-09
Vinyl Chloride	0.000116	0.012244898	1.42041E-06	1.9	2.69878E-06
			Pathway Cancer Risk		**8.95E-06**

Pathway: Inhalation of contaminated air

Chemical	Concentration	Intake	Final Intake	Slope Factor	Cancer Risk
Chloroethane	0.00039	0.030612245	1.19388E-05		ND
Chloromethane	0.0000972	0.030612245	2.97551E-06	0.006	3.86816E-08
1,2 Dichlorobenzene	0.0000521	0.030612245	1.5949E-06		ND
1,1 Dichloroethane	0.00149	0.030612245	4.56122E-05		ND
1,2 Dichloroethane	0.0000481	0.030612245	1.47245E-06	0.091	1.33993E-07
1,1 Dichloroethene	0.00084	0.030612245	2.57143E-05	1.2	0.000030857
1,2 Dichloroethene	0.000058	0.030612245	1.77551E-06		ND
Tetrachloroethene	0.0000855	0.030612245	2.61735E-06		ND
1,1,1 Trichloroethane	0.000578	0.030612245	1.76939E-05		ND
Trichloroethene	0.0000569	0.030612245	1.74184E-06	0.017	2.90E-08
Vinyl Chloride	0.000116	0.030612245	3.55102E-06	0.29	0.000001029
			Pathway Cancer Risk		**0.000032087**

FINAL INTAKE CALCULATIONS - Child, Cancer

Pathway: Ingestion of contaminated drinking water

Chemical	Concentration	Intake	Final Intake	Slope Factor	Cancer Risk
Chloroethane	0.00039	0.005714286	2.22857E-06		ND
Chloromethane	0.0000972	0.005714286	5.55429E-07	0.013	7.22057E-09
1,2 Dichlorobenzene	0.0000521	0.005714286	2.97714E-07		ND
1,1 Dichloroethane	0.00149	0.005714286	8.51429E-06		ND
1,2 Dichloroethane	0.0000481	0.005714286	2.74857E-07	0.091	2.5012E-08
1,1 Dichloroethene	0.00084	0.005714286	0.0000048	0.6	0.00000288
1,2 Dichloroethene	0.000058	0.005714286	3.31429E-07		ND
Tetrachloroethene	0.0000855	0.005714286	4.88571E-07		ND
1,1,1 Trichloroethane	0.000578	0.005714286	3.30286E-06		ND
Trichloroethene	0.0000569	0.005714286	3.25143E-07	0.011	3.57657E-09
Vinyl Chloride	0.000116	0.005714286	6.62857E-07	1.9	1.25943E-06
			Pathway Cancer Risk		**4.18E-06**

Pathway: Inhalation of contaminated air

Chemical	Concentration	Intake	Final Intake	Slope Factor	Cancer Risk
Chloroethane	0.00039	0.001185714	4.62429E-07		ND
Chloromethane	0.0000972	0.001185714	1.15251E-07	0.006	6.92E-10
1,2 Dichlorobenzene	0.0000521	0.001185714	6.17757E-08		ND
1,1 Dichloroethane	0.00149	0.001185714	1.76671E-06		ND
1,2 Dichloroethane	0.0000481	0.001185714	5.70329E-08	0.091	5.18999E-09
1,1 Dichloroethene	0.00084	0.001185714	0.000000996	1.2	1.20E-06
1,2 Dichloroethene	0.000058	0.001185714	6.87714E-08		ND
Tetrachloroethene	0.0000855	0.001185714	1.01379E-07		ND
1,1,1 Trichloroethane	0.000578	0.001185714	6.85343E-07		ND
Trichloroethene	0.0000569	0.001185714	6.74671E-08	0.017	1.00E-09
Vinyl Chloride	0.000116	0.001185714	1.37543E-07	0.29	3.90E-08
			Pathway Cancer Risk		**1.24E-06**

APPENDIX III

Risk Assessment Issue Paper for:

Provisional Oral RfD for Trichloroethylene

(CAS # 79-01-6)

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
OFFICE OF RESEARCH AND DEVELOPMENT
ENVIRONMENTAL CRITERIA AND ASSESSMENT OFFICE
CINCINNATI, OHIO 45268

RECEI.
APR 13 1992
REML SECTION

APR 10 1992

SUBJECT: Provisional oral RfD for trichloroethylene (CAS #79-01-6)

FROM: Joan S. Dollarhide Joan S Dollarhide
Associate Director
Superfund Health Risk Technical Support Center
Chemical Mixtures Assessment Branch

TO: Dave Crawford
U.S. EPA
Region VII

This memo contains our most recent assessment for a provisional oral RfD for trichloroethylene. Please note that the attached information has not been through the Agency's review process and therefore does not represent an Agency verified risk assessment.

If we can be of further assistance, please feel free to contact the Superfund Technical Support Center at FTS 684-7300.

Attachment

cc: J. Dinan (OS-230)
B. Means (OS-230)
K. Poirier (ECAO-Cin)
M. Williams (Region VII)

Attachment

Risk Assessment Issue Paper for:
Provisional Oral RfD for Trichloroethylene
(CAS # 79-01-6)

INTRODUCTION

An oral RfD is not available for trichloroethylene on IRIS (U.S. EPA, 1992a) or the HEAST (U.S. EPA, 1991a). The RfD/RfC status report (U.S. EPA, 1992b) states that the RfD is under review, but cites 7/22/85 as the last Work Group meeting concerning this RfD. OHEA documents listed on the CARA list (U.S. EPA, 1991c) include WQCDs (U.S. EPA, 1980), HADs (U.S. EPA, 1985; 1987a), and HEAs (U.S. EPA, 1984; 1988). None of these documents derived an oral RfD for trichloroethylene.

The Drinking Water list (U.S. EPA, 1991b) provides a value of 7E-3 mg/kg/day for the oral RfD for trichloroethylene with an associated DWEL of 0.3 mg/l; these toxicity values were derived in an ODW Health Advisory on trichloroethylene (U.S. EPA, 1987b). The basis for the RfD was a free-standing LOAEL for elevated liver weights in rats exposed to inhaled trichloroethylene for 14 weeks (Kimmerle and Eben, 1973). The derivation involved a determination of an absorbed dose for humans using the rat LOAEL, human inhalation rates and body weights, an absorption efficiency ratio of 0.3, and adjustments for continuous exposure. The absorbed dose (7.35 mg/kg/day) was divided by an uncertainty factor of 1000 (10 for the use of a LOAEL, 10 for interspecies extrapolation, and 10 for intraspecies variation).

ATSDR has prepared two Toxicological Profiles on trichloroethylene (ATSDR, 1989; 1991). The 1989 document derived an intermediate oral MRL of 2.2 E+0 mg/kg/day based on a NOAEL (217 mg/kg/day) for renal effects (increased urinary ketone and protein levels) in mice exposed to trichloroethylene in drinking water for six months (Tucker et al., 1982). The 1991 document derived an intermediate oral MRL of 1E-1 mg/kg/day based on a LOAEL of 100 mg/kg/day for increased liver weight in mice exposed by gavage for 4 weeks (Buben and O'Flaherty, 1985). Neither document derived a chronic oral MRL for trichloroethylene.

To identify research reports pertinent to the derivation of a chronic RfD for trichloroethylene, EPA and ATSDR documents on trichloroethylene (as cited above) and the HSDB, RTECS and TSCATS databases were reviewed; in addition, a computer search of the literature was conducted (TOXLINE, 1989 - January, 1992).

As reviewed by U.S. EPA (1985) and ATSDR (1989; 1991), trichloroethylene has been used as a surgical anesthetic, and effects on neurobehavior and the central nervous system are well studied in humans and animals exposed acutely to the inhaled compound. The effects of repeated exposures of humans to

trichloroethylene are less well studied. Occupational exposure to trichlorethylene in air has been associated with symptoms of effects on the central nervous system (e.g., nausea, headache, reduced cognitive performance, and sleep disturbances), but not on the kidney or liver (ATSDR, 1989, 1991; U.S. EPA, 1985; Nagaya et al., 1989; Ruijten et al., 1991). Data regarding effects in humans repeatedly exposed to trichloroethylene in drinking water are confounded by concurrent exposure to other chemicals (ATSDR, 1991; Goldberg et al., 1990). However, several studies are available in which animals have been repeatedly exposed to orally administered trichloroethylene. The data are reviewed herein, and a chronic RfD for trichloroethylene is derived.

CHRONIC ORAL TOXICITY

Nonneoplastic kidney lesions, in addition to carcinogenic responses, have been observed in studies designed to examine the carcinogenicity of chronic oral exposures to trichloroethylene in rodents.

NCI (1976) studied the carcinogenicity of trichloroethylene in corn oil in 78-week chronic gavage studies with rats and mice. The trichloroethylene sample used in these studies was ≥ 99.0% pure, but contained 0.09% epichlorohydrin, a demonstrated carcinogenic agent.

Groups of 50 male and 50 female rats were provided time-weighted average doses of 549 or 1,097 mg/kg/day (NCI, 1976). A matched vehicle control group contained 20 males and 20 females, and an unmatched vehicle control group contained an additional 79 male rats and 78 female rats. Rats were allowed to survive until 32 weeks after exposure. The exposed rat groups did not display statistically significant increases in incidences of tumors compared with control rats, but both exposed groups displayed decreased peak body weights and survival compared with controls. Nephropathy was common in both treated groups. The nephropathy was described as slight to moderate degenerative and regenerative changes in the tubular epithelium; the authors stated that these lesions were unlike those that frequently occur in aging Osborne-Mendel control rats.

Groups of 50 male and 50 female B6C3F1 mice were provided time-weighted average doses of 1,169 or 2,339 mg/kg/day for males and 869 or 1,739 mg/kg/day for females (NCI, 1976). A matched vehicle control group contained 20 males and 20 females, and an unmatched control group contained an additional 57 male and 60 female mice. Significantly reduced survival was observed in both exposed groups compared with matched vehicle controls. Significantly increased incidences of liver tumors were observed in both exposed groups of both sexes compared with the matched vehicle control groups. The occurrence of nonneoplastic lesions of the kidney were not mentioned in the report of this study.

In a second series of chronic gavage studies, NTP (1988, 1990) studied the carcinogenicity of epichlorohydrin-free trichloroethylene in rats and mice. The test chemical (designated as "Hi-Tri") used in these studies was tested to be > 99.9% pure and contained 8 ppm diisopropylamine as a stabilizer.

Trichloroethylene in corn oil was administered by gavage at doses of 0 or 1000 mg/kg to groups of 50 male and 50 female B6C3F1 mice for 5 days/week for up to 103 weeks (NTP, 1990). Adjustment for partial weekly exposures gives average daily doses of 0 and 714 mg/kg/day. Statistically significant differences between dosed and control mice included decreased survival in males, decreased body weights in male mice, increased hepatocellular carcinoma incidence in both sexes, increased adenoma incidence in male mice, and toxic nephrosis in both sexes. Toxic nephrosis, described as cytomegaly of the renal tubular cells, was observed in 45/50 male and 48/49 female dosed mice, but was absent in the vehicle controls.

Groups of 50 male and 50 female F344/N rats were administered gavage doses of 0, 500 or 1000 mg/kg trichloroethylene in corn oil for 5 days/week for up to 103 weeks (average daily doses of 0, 357, and 714 mg/kg/day) (NTP, 1990). Statistically significant differences between dosed and control rats included decreased survival of both low- and high-dose male rats, decreased body weights in both sexes of rats at both doses, increased incidence of renal tubular adenocarcinomas in male rats killed at the end of the study, and cytomegaly of the kidney. Renal cytomegaly was observed in 96/98 dosed male and 97/97 dosed female rats; no vehicle control rats displayed renal cytomegaly.

In another bioassay, groups of 50 male and 50 female rats of four strains (ACI, August, Marshall, and Osborne-Mendel) were administered 0, 500 and 1000 mg/kg trichloroethylene in corn oil by gavage 5 days/week for 103 weeks (average daily doses were 0, 357 and 714 mg/kg/day) (NTP, 1988). Depressions in final body weights ≥ 10%, compared with controls, were observed in ACI, August and Osborne-Mendel male rats and Marshall female rats exposed to 1000 mg/kg; final body weight depression ≥ 10% were observed only in ACI males at the 500-mg/kg dose level. Survival was significantly reduced in 7 of the 16 dosed groups compared with respective control groups. Clinical signs of central nervous toxicity (sedation, loss of consciousness, tremors, convulsions, and hindlimb paralysis) were observed following dose administration in male and female rats of all strains. Significantly increased incidence of renal tubular cell adenomas or adenomacarcinomas were observed only in low-dose male Osborne-Mendel rats, and interstitial cell neoplasms of the testis were observed in dosed Marshall rats. Exposure to trichloroethylene caused renal tubular cell cytomegaly in 82-100% of all dosed rats. Toxic nephropathy, described as dilated tubules lined by elongated and flattened epithelial cells, was observed in 17%-80% of the animals in the dosed groups. Cytomegaly or toxic nephropathy were not observed in untreated or vehicle control

groups. NTP (1988) concluded that these studies were inadequate tests of the carcinogenicity of trichloroethylene because of deficiencies in study-conduct and decreased survival, but clearly demonstrated the nephrotoxicity of trichloroethylene. NTP (1988) also concluded that the cause of early mortality in the dosed rats was not known but could have been due to gavage-related trauma, anesthetic properties of the chemical, nephrotoxicity or a combination of these factors.

SUBCHRONIC AND NEAR SUBCHRONIC ORAL TOXICITY

NTP has published results from 13-week gavage studies with rats exposed to trichloroethylene (NTP, 1988, 1990) and mice (NTP, 1990). The test chemical in this series of experiments was the same as designated for the chronic NTP studies reviewed in the previous section.

Groups of 10 male F344/N rats were administered gavage doses of 0, 125, 250, 500, 1,000 or 2,000 mg/kg trichloroethylene in corn oil 5 days per week for 13 weeks (NTP, 1990). Adjusting for the partial weekly exposure protocol, average daily doses are 0, 89, 179, 357, 714, or 1429 mg/kg/day. Groups of 10 female rats received doses of 0, 62.5, 125, 250, 500 or 1,000 mg/kg by the same schedule. (Adjusted doses were 0, 45, 89, 179, 357, or 714 mg/kg/day.) All rats survived to the end of the exposure period and only male rats dosed with 2,000 mg/kg exhibited depressions of body weight gain > 10%. Organ weight data were not reported. Histopathological examinations of major organs and tissues from the high-dose and control groups revealed cytomegaly and karyomegaly of the renal tubular epithelial cells in 8/9 high-dose males and 5/10 high-dose females, but not in the controls. The lesions were graded as minimal or mild in males and equivocal to minimal in females; these minimal renal effects were diagnosed during a reevaluation of the tissues after observation of pronounced renal effects in the subsequent 2-year study. Pulmonary vasculitis was observed in 6/10 high-dose males and 6/10 high-dose females (compared with 1/10 male and 1/10 female control rats).

In a separate rat study (NTP, 1988), groups of 10 male ACI and 10 male August rats were administered gavage doses of 0, 125, 250, 500, 1,000 or 2,000 mg/kg trichloroethylene in corn oil 5 days per week for 13 weeks (adjusted doses of 0, 89, 179, 357, 714, or 1429 mg/kg/day); groups of 10 females of these strains received doses of 0, 62.5, 125, 250, 500 or 1,000 mg/kg (adjusted doses of 0, 45, 89, 179, 357, or 714 mg/kg/day). Groups of 10 male Marshall rats received doses of 268, 308, 495, 932, or 1834 mg/kg by the same schedule (0, 191, 220, 354, 666, or 1310 mg/kg/day, adjusted doses); groups of 10 female Marshall rats received 0, 134, 153, 248, 466 or 918 mg/kg (0, 96, 109, 177, 333, 656 mg/kg/day, adjusted doses). All rats survived to the end of the study with the exception of 3 high-dose male August rats. Average depressions in final body weight > 10% (relative

to control values) were observed only in the high-dose male groups. Organ weight data were not reported. No clinical signs of central nervous system toxicity were recorded, and histological examination of major tissues and organs from high-dose rats did not reveal alterations compared with control tissues.

In the final NTP subchronic study (NTP, 1990), gavage doses of 0, 375, 750, 1500, 3000 or 6000 mg/kg were administered to groups of 10 male and 10 female B6C3F1 mice 5 days per week for 13 weeks (0, 268, 536, 1071, 2143, or 4286 mg/kg/day, adjusted doses). Deaths occurred in 2/10 males and 1/10 females at 1500 mg/kg, 7/10 males and 1/10 females at 3000 mg/kg, and all male and 9/10 females at 6000 mg/kg. Depressions in mean body weights were > 10% relative to controls in male mice receiving doses ≥ 750 mg/kg; body weight alterations were not apparent in female mice. Liver weight elevations (both absolute and relative) > 10% relative to controls were observed in male mice at doses ≥ 750 mg/kg and in females at doses ≥ 1500 mg/kg. Centrilobular necrosis was observed in 6/10 males and 1/10 females exposed to 6000 mg/kg. At the 3000 mg/kg level centrilobular necrosis was not observed in either sex, but 2/10 males had multifocal areas of calcification in their livers. Histopathological examinations of tissues from mice treated with the 3 lowest doses were not conducted. Mild to moderate cytomegaly and karyomegaly of the renal tubular epithelial cells was observed in all of the mice that received the two highest doses and survived for more than 6 weeks.

Stott et al. (1982) administered gavage doses of trichloroethylene (> 99.9% pure, stabilized with diisopropylamine) in corn oil at levels of 0, 250, 500, 1200 or 2400 mg/kg, 5 days/week for 3 weeks to groups of 10-12 male B6C3F1 mice. Adjusting for the partial weekly exposures gives average daily doses of 0, 179, 357, 857, or 1714 mg/kg/day. No exposure-related effects were observed on body weight, kidney weight or kidney histopathology. Increased relative liver weights and decreased DNA content per gram of hepatic tissue were observed at doses ≥ 500 mg/kg. Histopathological changes in hepatic tissues were observed at all dose levels. The severity of the changes increased with increasing dosage level. Slight increases in cytoplasmic eosinophilic staining of the centrilobular hepatocytes were observed at 250 and 500 mg/kg. At 1200 mg/kg increased centrilobular hepatocellular swelling was observed, and at 2400 mg/kg, more severe hepatocellular swelling, giant cell inflammation and mineralized cells were observed. Under the conditions of this study, the lowest dosage level of 250 mg/kg (179 mg/kg/day) was the LOAEL for response of the liver to trichloroethylene.

Stott et al. (1982) also administered gavage doses of trichloroethylene in corn oil of 0 or 1100 mg/kg, 5 days per week for 3 weeks, to groups of 4 male Osborne-Mendel rats. No treatment-related alterations in body weight, kidney weight,

histopathology of the kidney or liver, or DNA content per gram of renal or hepatic tissue were observed. Increased relative liver weight was the only significant treatment-related change observed in this study.

Tucker et al. (1982) provided trichloroethylene (reagent grade containing 0.004% diisopropylamine as stabilizer) in drinking water containing 1% emulphor at concentrations of 0, 0.1, 1.0, 2.5 and 5.0 mg/mL to groups of 30 male and 30 female CD-1 mice for 4 or 6 months. Average dosage levels estimated from water consumption data were reported to be 0, 18.4, 216.7, 393.0, and 660.2 mg/kg/day for males and 0, 17.9, 193.0, 437.1, and 793.3 mg/kg/day for females. No significant effects on weight gain were observed in the treated groups compared with the control group. The results of gross pathological examination of tissues at 4 and 6 months were reported to be unremarkable. Microscopic examinations of tissues and organs were not performed. Terminal body weights of male and female mice treated with the highest concentration of trichloroethylene were significantly decreased compared with the vehicle control terminal body weights. Increased relative liver weights were observed in males at both exposure times at the three higher doses and in females at the highest dose. Significantly increased kidney weights were observed in high-dose males at 4 and 6 months and in high-dose females at 6 months; urinalysis at 6 months of exposure showed elevated protein and ketone levels in high-dose females and males treated with the two highest concentrations of trichloroethylene. The NOEL of 0.1 mg/mL (18.4 mg/kg/day) and LOAEL of 1.0 mg/mL (216.7 mg/kg/day) for increased relative liver weight in mice describes the most sensitive toxicity threshold identified in this study. The LOAEL for kidney effects was 2.5 mg/mL (393 mg/kg/day).

In a study restricted to the hepatotoxicity of trichloroethylene, male Swiss-Cox mice (age 3-5 months, body weight 34-45 g) were administered distilled trichloroethylene (% purity not reported) in corn oil by gavage in doses of 0, 100, 200, 400, 800, 1600, 2400 or 3200 mg/kg on five days a week for 6 weeks (Buben and O'Flaherty, 1985). Adjusting for the partial weekly exposure gives average daily dosages of 71.4, 142.9, 285.7, 571.4, 1142.9, 1714.3 and 2285.7 mg/kg/day. Twelve mice per dosage were tested except for 5 mice at 100 mg/kg/day, 4 mice at 3200 mg/kg/day and 24 mice in the control group. The following endpoints were assessed on the day following treatment at all dosages: relative liver weight, liver glucose-6-phosphatase (G6P) activity, concentrations of liver triglycerides, serum glutamate-pyruvate transaminase (SGPT) activity. Liver DNA concentration and histology were evaluated at 285.7 and 1142.9 mg/kg/day. Statistically significant ($p < 0.05$) increases in relative liver weight at $\geq$ 71.4 mg/kg/day, G6P at $\geq$ 571.4 mg/kg/day, and SGPT at $\geq$1714.3 mg/kg/day were observed. The changes in relative liver weight and G6P were clearly dose-related. Liver triglycerides were significantly increased only at 1714.3 mg/kg/day ($p<0.01$); a comparable

increase occurred at 2285.7 mg/kg/day but was not statistically significant, apparently due to the small number of animals (4). The increases in liver size were attributed to hepatocellular hypertrophy based on histology and decreased hepatic DNA concentrations. Other hepatic histologic effects included degeneration, karyorrhexis (disintegration of the nucleus) and polyploidy at 285.7 and 1142.9 mg/kg/day, and necrosis at 1142.9 mg/kg/day. The degeneration was manifested by swollen hepatocytes that were not due simply to edema, as liver wet weight/dry weight ratios did not increase. Under the conditions of this experiment, the lowest dosage level (71.4 mg/kg/day) was a LOAEL for a dose-related response of the mouse liver to trichloroethylene which caused hepatocellular hypertrophy, and progressing to hepatocellular necrosis.

REPRODUCTIVE AND DEVELOPMENTAL TOXICITY

In a 2-generation fertility study (NTP, 1986), groups of 20 F_0 breeding pairs of F344 rats (11 weeks of age at the start) were provided diets containing nominal trichloroethylene concentrations of 0.15, 0.30 and 0.60% for a 7-day mating period, a 98-day cohabitation period, and a subsequent 28-day segregation period. A control group of 40 F_0 breeding pairs was provided a normal diet for the same period of time. Trichloroethylene (designated as "Hi-Tri Purity grade") was microencapsulated in a gelatin/sorbitol shell. Estimated average dosage levels were calculated from initial and week 13 body weight data reported by the authors and the allometric equation recommended by the U.S. EPA (1987c) for calculating food consumption by laboratory mammals. The estimated doses for male F_0 rats were 0, 130.2, 261.1, and 523.9 mg/kg/day; for F_0 females the doses were 0, 147.8, 301.7, and 599.3 mg/kg/day.

Statistically significant ($p < 0.05$) differences between the dosed and control F_0 groups were not observed in the following parameters: the proportion of breeding pairs able to produce at least one litter, the number of live litters per pair, the number of live pups per litter, the proportion of pups born alive, the sex of pups born alive (NTP, 1986). Dam body weights on postnatal day 0 were significantly depressed in all of the exposed F_0 groups compared with the control. Statistically significant ($p < 0.05$) trends with increasing dose were observed for decreased numbers of live litters per pair and for decreased numbers of live pups per litter. A crossover mating trial was subsequently conducted using three combinations of F_0 breeding pairs (20 pairs per combination) as follows: control male x control female; 0.6% male x control female; and control male x 0.6% female. In this trial, the only significant differences between the mating pairs with exposed partners and the control pairs were decreased proportion of detected matings (observed when either the male or female partners were exposed), and decreased bodyweight of the 0.6% dams on postnatal day 0. Exposure of either the male or female partner had no significant

effect on the other indices of fertility and reproductive performance listed above for the initial F_0 breeding trial.

Continuous exposure of F_1 rats (81 days $\pm$ 10) to the same dietary concentrations of trichloroethylene fed to their parents (14-20 breeding pairs were evaluated for each exposure level) had no effect on indices of mating, fertility or reproductive performance (NTP, 1986). As in the F_0 generation, treated F_1 dams displayed depressed body weight on postnatal day 0, indicating generalized maternal toxicity. Microscopic examination of major tissues and organs revealed no treatment-related pathological changes in either sex in the F_0 or the F_1 generations. At necropsy, body weights were depressed and liver weights (adjusted for body weight by an analysis of covariance) were increased in male and female F_0 rats treated with 0.6% trichloroethylene compared with control F_0 rats. F_1 male and female rats from all treatment groups displayed significantly decreased body weights at 21 and 81 (necropsy) days after birth. Significantly increased adjusted liver weights were observed for all treated F_1 male groups and for F_1 female rats treated with 0.3 or 0.6% trichloroethylene. Under the conditions of this experiment, the lowest exposure level (0.15% trichloroethylene) was a LOAEL for maternal toxicity demonstrated by decreased body weight (147.8 mg/kg/day), for decreased body weight and increased liver weight in F_1 males (130.2 mg/kg/day), and for decreased body weight in F_1 females (147.8 mg/kg/day).

In a similarly designed mouse study, NTP (1985) provided nominal concentrations of 0, 0.15, 0.30 and 0.60% trichloroethylene ("Hi-Tri Purity grade") in the diet of groups of breeding pairs of CD-1 mice starting at 11 weeks of age and continuing as described for the rat fertility study (NTP, 1986). The groups contained 35, 17, 18, and 19 pairs of mice, respectively. Average doses, in units of mg/kg/day, were reported to be 0, 63.8, 247.5, for week 1, 0, 52.5, 266.5, and 615.0 for week 2, and 0, 187.5, 375.0, and 750.0 for the remainder of the 18-week exposure period. Time-weighted average doses are calculated to be 0, 173, 362, and 737 mg/kg/day. No clinical signs of toxicity were observed throughout the exposure period. Indices of fertility and reproductive performance for the F_0 generation were not affected by exposure, except for a slight (< 10%), but statistically significant ($p < 0.05$), depression of birth body weights of live male pups or combined male and female pups compared with controls. The depression was only significant when adjustments were made for the total number of live and dead pups per litter by an analysis of variance.

Litters from the control and high-dose mouse groups were raised to sexual maturity to assess fertility and reproductive performance. Perinatal mortality was pronounced in the 0.6% group; a 61.3% mortality rate was observed compared with a 28.3% mortality rate for the control group. Survival after weaning was the same for both control and exposed F_1 groups. Surviving F_1 mice were provided the same feed level of trichloroethylene as

their parents for 74 ± 10 days; breeding pairs were then established and the F_1 females were allowed to deliver their litters. Indices of mating, fertility or reproductive performance for the 0.6% F_1 group were not significantly different from those for the control group.

Tissues from the control and high-dose F_0 and F_1 mice were weighed and examined microscopically (approximately 18 and 15 weeks of exposure for the F_0 and F_1 generations, respectively). Body weights at necropsy were not affected by high-dose exposure in either generation. Liver weights (absolute and adjusted) were increased by high-dose exposure in both sexes of both generations. Liver and kidney lesions (hypertrophy of the centrilobular liver cells and tubular degeneration and karyomegaly of the renal tubular epithelium) were also observed in high-dose F_0 and F_1 mice of both sexes. Significantly decreased proportions of sperm that were motile were observed in high-dose F_0 and F_1 males (45 and 18% decreases compared with controls). In summary, although trichloroethylene treatment at dietary concentrations as high as 0.6% did not alter several indices of fertility or reproductive performance, organ-specific effects on the F_0 and F_1 male reproductive tract and increased perinatal mortality of F_1 pups were observed. The authors concluded that trichloroethylene may present a selective risk to the neonatal mouse (NTP, 1985). The study identified 0.6% (737 mg/kg/day) as a FEL for the effects on the male mouse reproductive tract and neonatal survival, but did not identify a NOEL or NOAEL for these effects (neither endpoints were assessed at the lower exposure levels).

Manson et al. (1984) administered gavage doses of 0, 10, 100 or 1000 mg/kg trichloroethylene in corn oil to groups of 23 female Long-Evans hooded rats. Exposure commenced 2 weeks before mating, continued throughout mating (1 week), and was stopped on day 21 of pregnancy. Doses were administered 5 days/week for the first 3 weeks and 7 days/weeks for the last 3 weeks. Adjusting for the partial weekly exposure during the first part of the study, average daily doses were 0, 8.6, 85.7, or 857.1 mg/kg/day. Females were bred to untreated males. Indices of fertility (i.e., the average number of mating trials required for insemination and the number of rats which became pregnant) were not affected by exposure to any level of trichloroethylene. Maternal body weight gain during pregnancy, litter size at birth, and neonatal survival (up to 31 days after birth) were not altered in the groups exposed to 10 or 100 mg/kg. Body weight gains during the premating period and during pregnancy were significantly depressed only in the high-dose dams, as was decreased neonatal survival up to 18 days after birth (16.9% of 1000-mg/kg pups died compared with 7.7% in the control). Four deaths occurred among the 23 dams exposed to 1000 mg/kg. No major malformations were revealed by gross examinations of the pups. The authors speculated that the decreased neonatal survival was related to maternal toxicity rather than to specific

developmental toxicity. Under the conditions of this study, 100 mg/kg (85.7 mg/kg/day) was the NOAEL, and 1000 mg/kg/day (857.1 mg/kg/day) was the LOAEL for maternal toxicity and FEL for decreased neonatal survival.

DERIVATION OF A PROVISIONAL RfD

The chronic and subchronic mouse and rat gavage bioassays conducted by NCI (1976) and NTP (1988, 1990) identify the kidney (in mice and rats) and the liver (in mice) as target organs for trichloroethylene-induced nonneoplastic effects, however the data are not suitable bases for an RfD. The lowest doses in the chronic studies produced reduced survival, and, as FELs, cannot be used to derive an RfD. Deficiencies in the design of the subchronic NTP (1988, 1990) studies compromise their usefulness; histological examinations were conducted only on high-dose animals and controls, and organ weight data was reported for only one of the studies. In general, the NTP studies provide insufficient information for exposure to doses less than 500 mg/kg, a level identified as producing frank effects; the only exception is the mouse subchronic study (NTP) which identified 375 mg/kg (268 mg/kg/day adjusted for partial weekly exposure) as a NOAEL and 675 mg/kg as the LOAEL for increased liver weight in male mice. Other subchronic studies are available that identified LOAELs lower than 268 mg/kg/day (NTP, 1986; Tucker et al., 1982; Buben and O'Flaherty, 1985) .

The 2-generation fertility study of B6C3F1 mice (NTP, 1985) indicated that reduced neonatal survival during lactation is a significant effect produced by exposure to trichloroethylene. However, the study did not identify a NOAEL for this frank effect, and thus the data cannot be used to derive an RfD.

The 2-generation fertility study of F344 rats exposed to trichloroethylene in the diet (NTP, 1986) identified a free-standing LOAEL of 130.2 mg/kg/day for decreased body weight and increased liver weight in F_1 male rats exposed for 18 weeks to trichloroethylene; indices of fertility and reproductive performance and histological features of major organs and tissues in rats exposed to this dose or higher doses were not significantly different from comparable endpoints in controls.

While the 1986 NTP study is suitable for consideration as a basis for the RfD, the 6-month drinking water study of mice by Tucker et al. (1982) provides a better basis because it identified both NOAELs and LOAELs for the responses of the liver and kidney to orally administered trichloroethylene. The threshold for liver toxicity (NOAEL of 18.4 and LOAEL of 216.7 mg/kg/day for increased relative liver weight) was lower than that for renal effects (NOAEL of 216.7 and LOAEL of 393.0 mg/kg/day for elevated levels of protein and ketones; increased kidney weight was observed at the highest dose, 660.2 mg/kg/day).

Although the Tucker et al. (1982) study did not include histological examinations of the liver and kidney, a more comprehensive examination of hepatotoxicity in mice orally exposed to trichloroethylene for 6 weeks showed that liver weight increases were attributable to hypertrophy of the liver cells and that the hepatic response progressed to degenerative changes at higher doses (Buben and O'Flaherty, 1985). The study by Tucker et al. (1982) is a better basis for derivation of the RfD than the study by Buben and O'Flaherty (1985) because a NOAEL was identified and the duration of exposure was closer to a life-time.

A provisional chronic RfD of 6E-3 mg/kg/day is derived by dividing the mouse NOAEL of 18.4 mg/kg/day from the study by Tucker et al. (1982) by an uncertainty factor of 3000 (10 for interspecies extrapolation, 10 for intraspecies variation, 10 for extrapolation to chronic duration and 3 for weakness of the data base).

Confidence in the principal study is low. Adequate numbers of animals were exposed by a relevant route and were evaluated for several endpoints. However, histological examinations were not conducted on the tissues, and the duration of exposure was only one-quarter of a life-time. Confidence in the data base is low. Several subchronic toxicity studies in rats and mice are available, as are studies of reproductive performance in rats and mice. However, chronic oral bioassays do not adequately describe dose-response relationships for chronic oral exposure to low doses of trichloroethylene and comprehensive developmental toxicity studies are not available. Reflecting low confidence in the principal study and the data base, confidence in the provisional RfD for trichloroethylene is low.

REFERENCES

ATSDR (Agency for Toxic Substances and Disease Registry). 1989. Toxicological Profile for Trichloroethylene. U.S. Public Health Service. PB/90/127523/AS.

ATSDR (Agency for Toxic Substances and Disease Registry). 1991. Update Toxicological Profile for Trichloroethylene. U.S. Public Health Service. Draft for Public Comment.

Buben, J.A. and E.J. O'Flaherty. 1985. Delineation of the role of metabolism in the hepatotoxicity of trichloroethylene and perchloroethylene: a dose-effect study. Toxicol. Appl. Pharmacol. 78: 105-122.

Goldberg, S.J., M.D. Lebowitz, E.J. Graver and S. Hicks. 1990. An association of human congenital cardiac malformations and drinking water contaminants. J. Am. Coll. Cardiol. 16: 155-164.

Kimmerle, G. and A. Eben. 1973. Metabolism, excretion and toxicology of trichloroethylene after inhalation. 1. Experimental exposure on rats. Arch. Toxicol. 30: 115-126. Cited in U.S. EPA, 1987.

Manson, J.M., M. Murphy, N. Richdale and M.K. Smith. 1984. Effects of oral exposure to trichloroethylene on female reproductive function. Toxicology 32: 229-242.

Nagaya, T., N. Ishikawa and H. Hata. 1989. Urinary total protein and ß-2-microglobulin in workers exposed to trichloroethylene. Environ. Res. 50: 86-92.

NCI (National Cancer Institute). 1976. Bioassay of trichloroethylene for possible carcinogenicity. U.S. Department of Health Education and Welfare, Bethesda, MD. NCI-CG-TR-2. DHEW Publication No. (NIH) 76-802.

NTP (National Toxicology Program). 1985. Trichloroethylene: reproduction and fertility assessment in CD-1 mice when administered in the feed. Final report. National Toxicology Program, Research Triangle Park, NC. NTIS No. PB86-173150.

NTP (National Toxicology Program). 1986. Trichloroethylene: reproduction and fertility assessment in F344 rats when administered in the feed. Final report. National Toxicology Program, Research Triangle Park, NC. NTIS No. PB86-190782.

NTP (National Toxicology Program). 1988. Toxicology and carcinogenesis studies of trichloroethylene (CAS No. 79-01-6) in four strains of rats (ACI, August, Marshall, Osborne-Mendel). Gavage Studies. U.S. Department of Health and Human Services, National Institutes of Health, Bethesda, MD. NTR TR 273. NIH Publication No. 88-2529.

NTP (National Toxicology Program). 1990. Carcinogenesis studies of trichloroethylene (without epichlorohydrin) (CAS No. 79-01-6) in F344/N rats and B6C3F1 mice (Gavage studies). U.S. Department of Health and Human Services, National Institutes of Health, Bethesda, MD. NTR TR 243. NIH Publication No. 90-1779.

Ruijten, M.W.M.M., M.M. Verberk and H.J.A. Salle. 1991. Nerve function in workers with long term exposure to trichloroethene. Br. J. Indust. Med. 48: 87-92.

Stott, W.T., J.F. Quast and P.G. Watanabe. 1982. The pharmacokinetics and macromolecular interactions of trichloroethylene in mice and rats. Toxicol. Appl. Pharmacol. 62: 137-151.

Tucker, A.N., V.M. Sanders, D.W. Barnes et al. 1982. Toxicology of trichloroethylene in the mouse. Toxicol. Appl. Pharmacol. 62: 351-357.

U.S. EPA. 1980. Ambient Water Quality Criteria for Trichloroethylene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Water Regulations and Standards, Washington, DC. NTIS PB81-117871.

U.S. EPA. 1984. Health Effects Assessment for Trichloroethylene. Prepared by the Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH for the Office of Solid Waste and Emergency Response, Washington, DC. NTIS PB86-134574/AS.

U.S. EPA. 1985. Health Assessment Document for Trichloroethylene. Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC. EPA 600/8-82-006F. NTIS PB85-249696/AS.

U.S. EPA. 1987a. Health Assessment Document for Trichloroethylene. Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC. EPA 600/8-82-006FA. NTIS PB87-228045/AS.

U.S. EPA. 1987b. Trichloroethylene Health Advisory. Office of Drinking Water, Washington, DC. March 31, 1987.

U.S. EPA. 1987c. Recommendations for and Documentation of Biological Values for Use in Risk Assessment. Prepared by Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH for the Office of Solid Waste and Emergency Response, Washington, DC.

U.S. EPA. 1988. Updated Health Effects Assessment for Trichloroethylene. Prepared by the Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH for the Office of Solid Waste and Emergency Response, Washington, DC. NTIS PB90-142498/AS.

U.S. EPA. 1991a. Health Effects Assessment Summary Tables. Annual FY-1991. Office of Research and Development, Office of Emergency and Remedial Response, Washington, DC. NTIS PB91-921100.

U.S. EPA. 1991b. Drinking Water Regulations and Health Advisories. Office of Drinking Water, Washington, DC.

U.S. EPA. 1991c. Office of Health and Environmental Assessment Chemical Assessments and Related Activities. Office of Health and Environmental Assessment, Washington, DC. April, 1991. OHEA-I-127.

U.S. EPA. 1992a. Integrated Risk Information System (IRIS). Online. Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH.

U.S. EPA. 1992b. Monthly status report of RfD/RfC Work Group (As of 1/01/92). Office of Research and Development. Environmental Criteria and Assessment Office, Cincinnati, OH.